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VOL. X. JANUARY, 1930. No. 39

The Journal of Neurology and Psychopathology

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ISSUED BY THE BRITISH MEDICAL ASSOCIATION

London: British Medical Association House,

Tavistock Square, W.C. 1.

Published Quarterly. Subscription 30/- per annum. Single Numbers 8/6 net. Post Free.

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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY.

Vol. X. JANUARY, 1930. No. 39.

Original Papers.

CONGENITAL AUDITORY IMPERCEPTION (CONGENITAL WORD-DEAFNESS): AND ITS RELATION TO IDIOGLOSSIA AND OTHER SPEECH DEFECTS.*

By

C. WORSTER-DROUGHT AND I. M. ALLEN, LONDON.

In a previous communication[1] we described in detail a case of 'Congenital Auditory Imperception' in which word-meaning-deafness was the outstanding and important element. It was then pointed out that the disorganisation of the speech mechanism resulting from the auditory defect had led to a method of vocal expression of the nature of so-called 'idioglossia,' an individual language consisting of an extreme form of mispronounced and ill-expressed conventional language. In a further communication[2] the results of the examination of the patient by the methods employed by Head[3] in the investigation of aphasia were detailed and the findings described. As regards the latter, the conclusion was reached that in spite of the fact that such methods revealed more clearly the fundamental nature of the defect, they were not so helpful in the investigation of cases of congenital origin as in cases of aphasia which occur in adult life. The standpoint from which the findings were to be considered was also discussed and the opinion expressed that in congenital cases it might be more valuable to consider what the patient had acquired in spite of his disability, rather than the functions in which he was deficient on account of the defect.

In the present communication, the condition known as congenital word-deafness is reviewed, the clinical features and consequences of the defect are described, and its relation to speech defects and especially to 'idioglossia' is discussed.

NOMENCLATURE.

It is clear that when the earlier cases of congenital word-deafness were reported the association of the auditory defect with defects in speech was not

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fully understood. The descriptive terms employed were those used to describe the speech defects only and gave no hint of the presence of a defect in the field of audition. Wilbur[4] in 1867 called the condition 'aphasia,' and both he and Ireland[5] termed their patients "idiotic aphasics." Hun[6], Hall[7] and Crane[8] referred to the condition as an 'atavistic speech' or 'a sport in language.' Sainsbury[9] discussed the defect as a difficulty in speech, Hadden[10] described his cases under defects of articulation in children, and Coen,[11, 12, 13] also discussing the speech defect in particular, referred to it as 'Hörstummheit.' Hale White and Golding Bird[14] introduced the term 'idioglossia' to describe the curious form of congenital speech defect described by Hadden[10] and occurring in cases of their own, because the utterances of the patient gave the impression that he was speaking in a language of his own. Guthrie[15] defended the use of the term 'idioglossia' to describe the speech defect on the grounds that the speech of the patient at least produced the effect of an individual language, even though its abnormalities were due to the mispronunciation of ordinary English. He recognised that the condition was probably due to a congenital deficiency of audition not amounting to deafness but he still regarded the speech defect as the important feature of the condition and continued to use the term 'idioglossia' accordingly. Wyllie[16] used the term 'lalling' from the German word 'lallen' (to speak as a child), but, as Guthrie[15] pointed out, the use of this term was by no means justified.

Kerr[17] described his case as 'congenital pure word-deafness,' thus apparently assuming that there were no defects elsewhere. Syme[18] used the term 'congenital aphasia,' but thought that the condition was analogous to congenital word-blindness and that the term 'congenital word-deafness' would describe it more accurately. Gee[19], Ormerod[20], Town[21], McCready[22] and McCall[23] used the term 'congenital aphasia.' McCready[22] believed that this term was applicable equally to congenital word-blindness and congenital word-deafness; he recognised that objections had been raised to the use of all three terms but felt that as they had become sanctioned by usage, they were more likely to survive than other terms introduced to describe the conditions. In recording her cases, McCall[23] used the term 'congenital aphasia,' but included under it a case of congenital word-blindness as well as one of congenital word-deafness.

Thomas[25] discussed examples of the defect under the aphasias of childhood as 'congenital word-deafness' but regarded idioglossia as a distinct defect due to deficiency of the motor speech centre. Yearsley[26] believed that the term 'congenital word-deafness' should be reserved for cases which fulfilled the following definition—"a case in which the sense of hearing being otherwise normal there is failure to appreciate the significance of sounds." In referring to McCall's case, Rutherford[27] said that a case is not one of aphasia if the term 'congenital word-deafness' used in connection with it is correct. He regarded the latter condition as an example of a defect in the receptive centres

for spoken words and believed that "to such conditions the term 'aphasia' carrying with it the significance of imperfection in some part of the central neuro-motor apparatus for language is quite inapplicable." Tait,[28] also, was somewhat dubious concerning the use of the term 'aphasia' in such cases, as it implied the loss of something which had been previously present and for that reason was scarcely applicable to a congenital defect. Burr[29] described the condition as 'congenital word-deafness' and criticised the use of the term 'aphasia' in such cases for the same reason. Levy[30] referred to the condition as 'alalie idiopathique de Coen' and was apparently unaware of the association between the speech defect and congenital word-deafness.

Brônner[31] described one of her cases as an example of "marked deficiency in auditory powers, shown by poor auditory memory and defective discrimination of sound"; but in discussing a second case applied no descriptive term to the condition. Hughlings Jackson[32] was of the opinion that there was a defect in auditory appreciation on a lower level than that affected in aphasia and that the term 'imperception' could be suitably applied. Head[33] agreed that there were certain clinical manifestations justifiably grouped under the term 'imperception,' in which the patient suffered from more than word-deafness—the disturbance belonging to a lower order of functional disintegration than aphasia or amnesia. Wallin[34] discussed the condition under the term 'word-deafness,' 'auditory aphasia' or 'verbal amnesia.'

It will be seen from the opinions expressed above that there has been no agreement among writers as to the most suitable term to describe the condition. Clinically, the speech defect is often the most prominent feature, but the fundamental defect is the inability to appreciate the significance of words (word-meaning deafness) and sometimes even of cruder sounds. Hence it would appear that a term descriptive of the fundamental defect is the most suitable, and for this purpose the terms 'congenital word-meaning-deafness' and even 'congenital word-deafness' seem to be too limited in scope. Certain aspects of the condition suggest that considerably more than a defective appreciation of the significance of words is present, and on this account Hughlings Jackson[32] and Head[33] were probably right in regarding the defect as upon a lower level than aphasia. Consequently, the term 'congenital auditory imperception' describes the primary condition more completely than 'congenital word-deafness.' The only difficulty in the use of this term is that 'word-deafness' has been sanctioned by usage in adult cases and will probably on that account continue to be used in congenital cases. Its use in congenital cases is justifiable only so long as it is remembered that something more than the auditory appreciation of words is defective, that the defect in perception may extend even to include cruder sounds, and that the term is used in a special sense in such cases. So far, little attention has been paid to the fact that the most obvious clinical feature of the condition is the speech defect. It is perfectly justifiable, however, to apply to the condition a descriptive term which draws attention to the primary defect only, even when,

as we have shown in a previous communication, it is understood that defects are present in speech, in the visual appreciation of symbols, and in writing. There is no more justification for the use of a term descriptive only of the defect in speech than there would be for the use of one describing only the defect in visual appreciation or that in writing. A term descriptive of the primary and fundamental defect should be sufficient and on the whole it is felt that 'congenital auditory imperception' is probably the best for the purpose. On account of the long-established use of 'word-deafness' in adult cases, however, the term 'congenital word-deafness' is used in its special sense in order to prevent confusion.

HISTORY.

The history of the subject of congenital word-deafness and its complications is a reflection of the different phases through which the subject has passed since aphasia was first recognised as a clinical abnormality early in the nineteenth century. Hence it follows that references to the subject are to be found under the headings of idiocy in children, speech defects (and especially idioglossia), congenital aphasia, sensory aphasia, congenital word-deafness, the association of congenital word-deafness and congenital aphasia with speech defects, behaviour defects, studies in psychology and educational problems.

In 1828 Gall differentiated the processes of memory into distinct 'faculties' in which he included those of 'word memory' and 'the sense of spoken language.' He described the variations in these functions in diseased conditions, and anticipated both the 'word-blindness' of Kussmaul and the 'motor aphasia' of Broca. In addition he described what McCready[21] regards as faulty speech memory in children and not only gave a clear clinical picture of 'congenital aphasia' but also thus early differentiated those who were feeble-minded from those who were mentally normal. In 1865 Benedikt (according to Kerr[17]) described an intelligent boy who heard and apparently understood but could say only 'papa' and 'mama.' In 1867 there appeared from the pen of Wilbur[4] a communication which indicated at least in some measure the attitude which at that time was assumed towards the then unrecognised defect. In a paper upon aphasia in the mentally afflicted he reviewed the speech defects in 443 idiots under his care and with reference to their powers of expression arranged them in nine classes, the most important of which from the point of view of the subject under discussion was that including "those who repeat a sentence after hearing it but have not the power to originate it." That at least some of them were examples of the condition now known as congenital word-deafness is suggested by a study of four of the cases quoted by him in his paper. The first case was a boy of thirteen who attempted to speak only one or two words which his father alone could understand, and "used his eyes more than his ears." He did not attempt to s[illegible] until the age of nine and after his education was undertaken he learned [illegible] read much more easily

than to speak. The second case was that of a boy of eight whose condition was described as "idiocy supervening in infancy." Though his mother declared that he spoke, it was found that he merely repeated what he heard, and never originated speech. His lack of spontaneous speech was thought to be due to "the absence of proper volition," but it is significant that the defect was eventually overcome by education through the visual channel. The third case was that of a boy of thirteen whose condition was described as "idiocy resulting from convulsions in infancy." It was noticed that if he were asked to repeat two or three words he would say only the last one—"ball" for "my ball," and "book" for "the book." It was remarked that "his memory would not go back to the last word in his attempt at repetition." The fourth case was that of a boy of eight who made no attempt to speak. It was noted that when shown a printed word he knew what it represented and pointed to the object "but his memory seemed at fault as to the name until it was pronounced in his hearing." It is significant that of the ten cases quoted by Wilbur[4], four were almost certainly cases of congenital word-deafness as the condition is now defined and that two others, but for the fact that they were described as partially or slightly deaf, might be placed in the same category. It is noteworthy also that all the cases were in an institution for idiots; from this it may be assumed, apart from the descriptions applied to the cases quoted above, that, like deaf mutes, children suffering from this disability were thought to be unable to understand spoken language because they were mentally deficient. In all four cases the descriptions reveal no defects other than those of hearing and speech and in fact stress the point that the children were well-behaved and intelligent in all other respects.

In 1868 Hun[5] described the case of a girl of four and a half, and that of her younger brother who "spoke a language of their own" thought to resemble French; and in 1886 Hall[7] recorded the cases of two twins of German extraction who spoke a language of their own thought to resemble German. These four cases were regarded by Guthrie[15] as cases of idioglossia, a feature of the condition which will be discussed at a later stage.

In 1889, Coen[11] of Vienna described under the name of 'Hörstummheit' a form of congenital inability to speak, occurring in children and especially in boys who were normal in body and mind and whose speech organs were apparently healthy. In the same year Sainsbury[9] described the case of a boy of five and a half who suffered from difficulty in speech to such an extent as to be unintelligible to strangers: he recorded the alphabet as pronounced by this patient as follows: "sa, p, see, t, e, sa, see, sa, sow, sa, sa, sow, sa, sa, sow, p, two, sa, sa, t, soo, fee, tow-see, sa, wow, sa." He then proceeded: "I may suggest that the speech error may be of the nature of a simple defect, i.e., fault by arrest, the centres in the cortex having failed to develop; or it may be of the nature of an actual perversion. In either case there may be simply an inability to reproduce the sound given, the child being conscious of the

imperfection of his own copy, or there may be a condition analogous to colour-blindness, a failure to recognise sound differences." The nature of the speech abnormality in this case suggests that it is the same as the 'idioglossia' described by Hadden[10] and others, a feature of congenital word-deafness which we have described according to Head's[3] terminology as 'verbal aphasia'; the explanation offered is interesting as it is the first suggestion on record that the speech defect is due to an inability to recognise differences in sound.

In 1891 Hale-White and Golding Bird[11] described under the name of 'idioglossia' a defective condition of speech which gave the impression of an individual language in two brothers aged ten and a half and nine years, respectively. This speech defect is mentioned by Bastian[35] as a congenital speech defect. "These children have to a certain extent a language of their own so that when asked to repeat phrases they make use of different, though definite, sounds instead of those proper to the words that should be employed. The sounds which they substitute are said to be always the same for the same words." This last peculiarity McCready[21] believed to be due to the fact that the child simply repeated the sounds as he heard them. In 1891 Taylor[36] described a case of a similar speech defect in a boy of eight and a half. In the same year Hadden[10] gave a full account of three cases in illustration of defects of articulation—all males and aged respectively eleven, seven and four years. In the first case the language used was similar to that described by Sainsbury[9] in his case; and "when asked to copy or write at dictation short and simple sentences, he would transcribe the first two or three words correctly, and then he would seem puzzled, the remaining words being represented by unmeaning up and down strokes or by combinations of letters like ve, va, etc., which recalled his spoken language. He soon became hopelessly confused and unable to proceed." In the second case the language was similar and "many other defects appeared when he attempted to combine sounds into words." In the third case "the power to combine the elementary sounds in ordinary articulate language was very defective and his speech was accordingly almost unintelligible." Hadden[10] suggested that the condition was almost certainly dependent upon some fault in the central nervous system, and thought it was due to a defect of a part of the brain "which co-ordinates the fifty or so elementary sounds into more complex forms." He appeared to discuss the defect entirely from the motor aspect and made no reference to any auditory defects. He suggested for such cases methods of education which had met with some success at his hands.

In 1895 Colman[37], in the course of a discussion on various speech defects, mentioned 'idioglossia' as an inability to pronounce certain letters and the substitution for them of totally dissimilar sounds. He described two cases in detail and mentioned that he had notes of six other cases of the same type. His first case was that of a boy of six who repeated the Lord's Prayer in the following way: "Oue tabde ne nah e nedde, anno de di na; i dede ta, i du

de di ou eeth a te e edde. Te ut te da oue dade de, e didde ap tetedde, a ne adiu to tetedde adase us, ne notte tetate, mime, utte enn, to i aitevene, pore e dande, to edde e edde. Ame." No mention was made of any defect in the understanding of words. In his second case, a boy of six, the speech was quite unintelligible.

In 1900 Kerr[17] published under the title of 'congenital pure word-deafness' a case which he subsequently claimed to be the first authoritative case of the condition published. This was a boy of nine who "was very intelligent, without discoverable defect, except that he could not understand words; he had good hearing but did not speak until taught articulation." Kerr[38] mentioned that he had recognised this case because he had seen cases of congenital word-blindness and expected to find a similar defect in the auditory field. In 1904, Syme[18] described the condition of a boy of eight who "when spoken to did not seem to hear" though his hearing was good, "had no idea of the meaning of words" and had never spoken in an intelligible manner. He suggested that in this case there was either a faulty development of the auditory centre or an interference with the afferent path to this centre, and rather inclined to the former view.

In 1907 Guthrie[15] reviewed the literature of idioglossia and described two cases of the condition, the first in a girl of seven and the second in a boy of seven and a half. He reproduced the alphabet as pronounced by his cases, drew attention to the familial factor in the condition and pointed out that it was not necessarily associated with mental deficiency. He concluded that there was no defect in the motor apparatus of speech, and that want of attention and mental concentration was largely concerned in perpetuating the faulty articulation. In connection with the case he described in detail, Guthrie was of the opinion that the defect was not in the motor vocal apparatus but in the ear; "it is not that he is deaf, but that he lacks the power of discriminating shades of difference between sounds." Guthrie defended the use of the term 'idioglossia' on the grounds that the speech of those affected produced the effect of a new language though it was actually nothing more than imperfectly pronounced English; he criticised the use of the term 'lalling' suggested by Wyllie[19] and adduced evidence to dispose of the suggestion that idioglossia was actually an atavistic language as suggested by Hall, Hun and Crane[8]; also he disproved the theory that it was a baby language. This author expressed the opinion that idioglossia is simply an exaggeration of defective modes of speech which are extremely common in minor degrees without being regarded as in any way morbid; and in discussing the part played by defective audition in producing idioglossia quoted Hadden's opinion that "the evolution of speech depends upon the integrity of the auditory perceptive centres" and that of Spencer Watson[39] and Langdon Brown[10] that the condition was due to a defect of hearing short of deafness.

In 1908 Thomas[25], in discussing the aphasias of childhood and educational hygiene, pointed out that individuals could be classified into 'auditives,'

'visuals' and 'motor-minded' according as they remembered and thought chiefly in auditory, visual or kinæsthetic terms: he also expressed the opinion that in every 'faculty' there could be found all kinds of variation from extreme facility to entire absence of organisation. Thomas then proceeded: "If this be true, we should find some children who are unable to organise their auditory word centres, although otherwise intelligent, and unable, though they hear, to store up memories of the meaning of words heard"—the congenitally word-deaf. He remarked that some examples of the condition were to be found in almost every class for the deaf, and that the common explanation of the condition was that "they do not understand because they are too lazy to do so." He described two cases in detail. The first was that of a boy of eight who had never attempted to talk. His hearing was normal to all the usual tests but he could not understand words; he repeated sentences after the speaker in an imperfect manner, such as "Det me a pentil" for "Get me a pencil"; and made an obvious attempt to understand a few words by keeping his eyes fixed upon the speaker's lips. His second case was that of a boy of four whose hearing was good, who made an attempt to repeat words heard but could attach no meaning to them, and by lip-reading understood the words seen and repeated them correctly. His voice and intonation were normal. Because of the prevailing theory based upon the work of those whom Head[33] called the 'diagrammists,' Thomas recognised no relationship between congenital word-deafness and idioglossia and expressed the opinion that the latter defect was due to imperfect organisation of the motor speech centre and that most minor speech defects in children were also due to the same cause.

In 1909 Yearsley[11] reported a case of "a peculiar form of (?) word-deafness" in a girl of eleven who had been at a deaf school for three years. This patient had "only vowel hearing and could produce no words"; she repeated words as they were spoken, had normal hearing to ordinary sounds and was normal in every other respect. In this case the defect was thought to be due to "weakness of the auditory word centre." In 1910 McCready[12] again suggested that idioglossia was due to partial word-deafness, that it resulted from the child's repeating words parrotwise as he heard them, and that the speech of those affected was intelligible only to those in close contact with them and to young children of the same age. In 1911 McCall[22] described a case of congenital word-deafness in a boy of eight who "when spoken to took no notice and might easily have been mistaken for a deaf child," but at once had his attention attracted by a noise. "His speech was markedly defective and the intonation resembled in a slight degree that peculiar to cleft palate. As words, however, meant nothing to such a child, he did not attempt to imitate them unless directly made to do so: he therefore did not learn to talk." In the same year Tait[28] described a case in a boy of five under the title of "congenital deficiency of speech areas (congenital aphasia ?)." This boy could not utter a single word, but phonated a sound like "ta" or "ah" suggestive of cleft

palate. His hearing was good and he was said to understand what was said to him, but in this case as in many others whose records we have examined, there was nothing in the report to indicate that any careful investigation had been made to show that there was a distinction between the understanding of words seen and those heard. In 1911 Levy[30] discussed the speech defect previously referred to as idioglossia under the title of 'entendants-muets' or 'alalie idiopathique de Coen.' He described it as a type of dumbness in children more than three years old, of normal or almost normal appearance, and without any deafness. He provided the following concise definition of the condition as he understood it: a type of speech defect presenting the following positive and negative characteristics—(1) children who are normal physically and mentally; (2) hearing normal; (3) the peripheral organs of speech properly formed and functioning normally; (4) absence of paralysis, atrophy or contracture of the trunk and limbs. He described the sufferers from this condition as children who hear well and have all the appearances of children of their age excepting that they do not speak at all or have a rudimentary language for their age. The cause of the defect he regarded as obscure. Levy mentioned five cases of the condition all of which he had observed for a period of five years, but apart from one who was described as a female of sixteen when first seen no details were given of the abnormalities.

In 1911 Town[21] gave a comprehensive historical review of congenital aphasia and described two cases of congenital word-deafness in her account of the condition. The first case was that of a boy of eight who could not understand or use language normally. He was not deaf, made his wants known by gestures and comprehended directions given by signs. "He could repeat words uttered in an ordinary tone, not correctly, but in a way that approximated to the original." He could name about thirty-six objects or their pictures and used voluntarily about twenty-four words, so that he had a vocabulary of about sixty words. "He heard other words, as was proved by the fact that he would repeat them, but he did not understand them, as was shown by his failure to comply with requests or point out objects." She also reported a second case the details of which are not available.

In 1912 Burr[29] discussed congenital aphasia and pointed out that no necropsy of a case of congenital cerebral speech defect leading to motor aphasia had ever been performed. He stressed the fact that all discussions upon aphasia had been based upon adult cases in which the main consideration was the comparison of the condition of the patient with that previous to the onset of the lesion giving rise to the aphasia, and mentioned that of the pathological variations of speech in children only deaf-mutism had been studied in any detail. He discussed the prevailing opinion that when a child was mute and not deaf he must be an imbecile, and sought in his paper to combat this impression. He summarised the arguments against the existence of a developmental aphasia as follows—(1) the absence of necropsies proving it, against

which there were no necropsies disproving it; (2) the fact that acquired aphasias in children are usually recovered from, presumably by education of the opposite cerebral hemisphere even when the lesion is destructive. From the latter, Burr concluded that a bilateral lesion would be necessary in congenital cases and the chances of this were small. He believed nevertheless that (1) there are individuals who, without congenital or early defect, never develop the ability to speak on account of disturbances confined to the speech centres, though hearing and the understanding of heard words are normal; and that (2) "there are still others who are congenitally word-deaf and whose mental deficiency depends upon the speech defect, the verbal amnesia or rather the inability to fix in memory the impressions of heard words, rather than that the speech defect is the result of the imbecility." He stated that, nevertheless, although their mental condition is potentially normal, "these cases without suitable environment will grow up imbeciles from deprivation as deaf-mutes formerly did." He stressed the fact that, in the absence of necropsies, a discussion of morbid anatomy would be of no assistance, and was of the opinion that even with them "it is entirely conceivable that the speech centres may on gross examination show no sign of disease and yet be so poor in well-developed cells as to be unable to perform their functions." Burr then described four cases to show that they were potentially normal in mind and with proper environment would attain to average mentality. His first case was that of a boy of twelve who had suffered from congenital syphilis and was entirely speechless and mute except for a few inarticulate sounds. On examination he showed definite evidence of congenital syphilis: in addition, it was noted that he apparently understood what was said to him but that with education his intelligence, or rather his knowledge, increased a great deal and that apart from speech he learned things quickly and well. The second case was that of a boy of three who had never made an effort to talk though he apparently heard perfectly well. The third case was that of a boy of seven who up to the age of five could repeat words immediately after he heard them, but after that could speak only a few words spontaneously and those never in sentences of more than three words. He could repeat a number of words on hearing them, but no new words could be fixed in his memory though his memory for events was good. Burr's fourth case was that of a girl of seventeen who was for a time in a school for the mute and had never spoken beyond saying the words 'yes' and 'no' which she usually but not always employed correctly. Although it was recorded that she could hear and understand spoken language, it was not stated how complete was the investigation upon which that opinion was based.

In 1913 Variot[43] described, under the general title of "congenital aphasia without motor or sensory abnormalities and with apparent preservation of normal mentality," the case of a boy of six who could not speak beyond saying a few monosyllables and bisyllables and the words "papa" and "merci,"

which he repeated parrotwise in a somewhat explosive manner and with the syllables spaced. He could neither read nor write but was recorded as being able to understand a few spoken words. Variot concluded that the case was an unusual one of congenital aphasia different from that occurring in imbeciles and perhaps associated with a defect in the centre for spoken language or with an undiscovered lesion which had occurred in early childhood. The statement that the patient "not only heard spoken sounds but also understood them" is rather against the inclusion of Variot's case as one of congenital word-deafness, but here again no details of the method of examination employed are given.

With the steady growth of interest in the psychological reactions to environment and difficulties, it is not surprising that the next communication upon the subject should be concerned with the abnormal psychological reactions which sometimes complicate the defect. In 1919 Brônner[31], in a discussion on the psychology of special abilities and disabilities, defined the term 'congenital word-deafness' as referring to the inability to understand and use spoken language, and pointed out that though the term was frequently employed there were few examples of the defect in the literature. She quoted Town's[21] first case and referred to her second case, believing that both could be explained more satisfactorily than as the result of a congenital defect. She then proceeded: "An essential peculiarity in regard to this problem is that tests proving the hearing normal at the time of examination by no means show that in the past there was no auditory defect such as would be caused by an inflammatory process, notably otitis media"; and seemed to think that lack of consideration of the temporary loss of hearing which might result from this condition lessened the value of the cases described by Thomas[25] and McCall[23]. Though it would appear to be improbable that temporary loss of hearing from such a condition could result in loss of the understanding of words spoken after recovery, a case described by Drinkwater[41] in 1911 suggested that there might be some slight reason for such an opinion. This was the case of a girl of fourteen who had been completely deaf for four years after the onset of an ordinary 'cold in the head.' When hearing returned after the removal of adenoids, it was observed that she could not understand what was said though she could hear a person speaking. Drinkwater suggested that the auditory word centre had probably become inactive owing to being unexercised for four years and the patient had forgotten the meaning of spoken words. It was noted, however, that the patient made no attempt to repeat words and sounds after the observer, a fact which suggests that the condition was still one of partial deafness rather than of word-deafness, for from our examination of the cases in the literature we have concluded that patients suffering from congenital word-deafness will and do repeat sounds and words after the observer in an imperfect manner. Brônner's[31] criticism of Town's[21] cases suffers from the fact that it is based upon the consideration of the psychological aspects of the

defect and pays little attention to the clinical aspects of the condition as revealed in the history of the patients. Having discussed this aspect of the problem, Brônner then stated that she had never studied a case of congenital word-deafness and immediately afterwards quoted the following two cases which in our opinion were true examples of the condition. The first was that of a boy of fifteen and a half who had been sent for investigation as a behaviour problem. He made errors in the reading of simple words; was certain only of some monosyllabic words; and could not spell correctly. "We found that the boy could not depend upon the sound of words at all; these evidently meant nothing to him. He himself said he had to learn his spelling entirely by 'the looks of words.' . . . He wrote 'gril' for 'girl' and 'print' for 'printer.' . . . He had no sense of the phonetic values. . . . On examination he showed defects in auditory memory. . . . His enunciation was extremely faulty though his vocabulary showed a fairly wide range. . . . It was evident that he did not hear all the sounds, for certain syllables were slurred over in every trial. . . . His auditory perception was exceedingly faulty and his auditory memory was poor." Furthermore, he was described as a case illustrating "marked deficiency in auditory powers, shown by poor auditory memory and defective discrimination of sounds." The second case was that of a boy of sixteen who illustrated a defect in several aspects of language. Although the clinical investigation was imperfect he appears to have been a true example of congenital word-deafness, as the following extracts from Brônner's description show. "We were impressed over and over again with the fact that words seemed to have no significance to the boy. He had the greatest trouble in expressing himself, nor could he control his verbal associations in tests requiring these. . . . He was unable to write a simple sentence from dictation and when he himself wished to write an account of his schooling the result was ludicrous. Even very simple words were misspelled, as is shown in the following—'I near wast one plaes atsed in Chicago' (I never went any place outside of Chicago)." In addition to these two cases, Brônner described two others which were apparently of a more limited type. The first was that of a boy of fifteen and a half who showed inability in number work based upon a "defect in auditory memory for numbers." The second case was that of a boy of sixteen and a half who, in spite of good educational advantages, showed a defect for number work which was not merely poor auditory memory but this combined with poor powers of forming associations with symbols. In reading he made errors with small words; he made errors in spelling from dictation; his control of verbal associations was not good, and his visual memory was better than the auditory.

In 1926 Head[33] pointed out that there were certain clinical manifestations which could be justifiably grouped under the terms 'auditory imperception' or 'agnosia,' and stressed the fact that the patient suffered from much more than word-deafness. The disturbance belonged to a lower order of functional

disintegration than aphasia and amnesia, for at one time or another the patient had difficulty in recognising the meaning of crude sounds. This was of necessity accompanied by some want of verbal appreciation or inability to read and understand printed symbols; but all such lack of comprehension of language was a secondary consequence of the gross imperception.

By 1927 the importance of the subject of congenital word-deafness, in spite of the comparative rarity of the condition, was fully appreciated and it was recognised as a factor to be considered in education. Wallin[31] defined it as a condition in which in severe cases spoken language is heard but not comprehended. "The difficulty, again, is not in the auditory perception of the spoken words, but in the recognition of the acoustic word imagery." He stressed the speech defects associated with and dependent upon the condition and, emphasising the rarity of congenital word-deafness, pointed out that he had met with only one case in an extensive clinical experience. This case was that of a boy of seven and a half who, though quite intelligent in other matters, was unable to do the lowest grade of school work and was most deficient in language and speech. He was two before he used single words and seven before he spoke in short phrases. He always watched the speaker's lips and facial expression though he took care to speak very loud. The words were heard but many of them seemed not to be understood. After studying him for several years, Wallin came to the conclusion that he was subnormal in general intelligence, and that he was subject to "congenital partial auditory aphasia with partial motor aphasia." He also pointed out that with congenital word-deafness there was sometimes associated "psychic or mental deafness" which he defined as "the loss of the ability to understand the meaning of or to recognize sounds and noises. The affected person hears the sound but cannot identify it and not being able to recognize it does not know how to react to it (sensory apraxia)." In addition, in musical persons 'amusia,' or "the loss of ability to apprehend melodies or to recognize musical sounds" was frequently associated.

INCIDENCE.

Congenital word-deafness is a comparatively rare defect and much more uncommon than the analogous condition, congenital word-blindness. In our review of the literature which has covered a period of approximately sixty years we found records of or references to only fifty cases which could be interpreted as examples of the condition. While in no way assuming that these are all the cases reported, they probably represent the majority of them and of the cases which have been described in sufficient detail for an opinion to be expressed upon them. Of necessity, certain of the cases included have had to be interpreted as such on incomplete evidence, and the difficulties have been increased considerably by the fact that the term 'congenital word-deafness' was apparently not in common use until about 1910. Although Sainsbury[9] and Coen[11] had in 1889 hinted at the nature of the condition, the first case to

be definitely diagnosed as such was that of Kerr[17] in 1900; but in 1907 Guthrie[15] described cases under the name of the associated speech defect—'idioglossia'—and in 1911 Levy[30] referred to cases as 'alalie idiopathique.' Thus it follows that references to the condition have been found under 'idiocy' before 1870, under 'speech defects' from that time till 1907, and after that under discussions of psychological and educational problems, while only a few of the cases quoted have been described under the term 'congenital word-deafness.' In some records no reference whatever has been made to the receptive functions; in others the method of examination employed has not been mentioned, and in others again the psychological method of approach alone has been employed. Consequently, it has been found necessary to form some conception of the symptomatology and the complications of the defect from our one case and from the more detailed of the descriptions in the literature of the past twenty years; and to consider every other case in the light of this conception. Upon this basis, only fifty cases have been collected from the literature over a period of sixty years, although there are descriptions of others, such as those of Coen[11] (1889) and Chazal[13] (1907), to which we have not been able to gain access.

Up till 1870 most of the cases of congenital aphasia reported were of the auditory type, but following the first description of a case of congenital word-blindness by Broadbent[16] in 1872, the number of cases of congenital word-blindness reported far exceeded those of congenital word-deafness. It is significant of the relative frequency of the two conditions that in McCready's paper[21] (1926) on defects in the zone of language (word-deafness and word-blindness) numerous cases of congenital word-blindness are referred to but not a single case of congenital word-deafness is mentioned. The contrast is more apparent if the frequency of congenital word-blindness as found by certain observers is compared with the number of cases of congenital word-deafness reported in the literature. Of the former condition Warburg[47] found 14 cases among 2,000 ordinary school children in Cologne, and seven cases among 400 children in special schools, while Thomas[48] estimated that in London schools the frequency of congenital word-blindness was about one in 2,000.

The rarity of congenital word-deafness has been referred to by other writers. When he described his case in 1904 Syme[18] claimed that no other case of the same type had been described, and that no post-mortem examination had ever been done on such a case. Burr[29] and Levy[30] also referred to the rarity of the condition. Burr pointed out that the literature on the subject was very meagre, the number of such children very small and post-mortem evidence of the cause of the condition non-existent; while Levy was unable to consider the cause of the defect because of lack of material. Other authors who have contrasted the relative frequency of congenital word-blindness with the comparative rarity of congenital word-deafness are Rutherford[27], Yearsley[26], Brônner[31], Thomas[25], Kerr[38] and Wallin[24]. Rutherford pointed out that congenital aphasia, by which he apparently meant congenital word-deafness,

was distinctly rare and was easily confused with deaf-mutism by the unwary; Yearsley said that all the suspected cases of the condition he had seen had, with one exception, proved to be other conditions; while Wallin had met with only one case in the course of an extensive clinical experience.

It is interesting to speculate upon the reasons for the comparative rarity of the defect and for the decided contrast between its rarity and the relative frequency of congenital word-blindness.

(1) The symptomatology of congenital word-deafness is not so obvious as that of congenital word-blindness. In the latter the child does not and cannot learn to read, whereas in the former the lack of understanding of spoken language may not be so obvious, and 'idiocy,' 'imbecility,' dumbness, speech defects and behaviour defects may be more prominent features of the condition. Cases of congenital word-blindness are more easily recognised, even by teachers, than are cases of congenital word-deafness.

As Thomas[25] has pointed out, there are probably children who can hear and yet not understand in every school for the deaf; while in France deaf-teachers have recognised the existence of a similar class, but have usually explained that the defect was due to the laziness or inattention of the pupil. Hence it follows that, because of the varied nature of the symptomatology and complications of the defect, such cases have often been accepted as examples of their most prominent feature and, in the absence of detailed investigations with the object of finding the underlying defect, have rarely been diagnosed correctly or completely. It is possible therefore that the incidence of congenital word-deafness in the general population is much greater than is appreciated from the small number of cases which has appeared in the literature.

(2) Though he recognised that children afflicted with congenital word-deafness are potentially normal both mentally and socially, Burr[22] pointed out that, without suitable environment, they are apt to become 'imbeciles from deprivation,' as deaf-mutes formerly did before the true nature of their condition was fully understood. Thomas pointed out that congenital word-deafness was of greater importance in the struggle for existence than congenital word-blindness and that sufferers from the defect were more liable to fail in their economic and social adjustments. Wilbur's[4] cases were in an institution for idiots and constituted a part of his class of idiots "who could repeat words but not originate them." Ireland[5], in writing on the care of mental defectives, said that there was a small class of 'idiotic aphasics' who remain obstinately mute "though it is clear that they have more intelligence than other children who talk volubly." Hence it is necessary, in view of the possibility of 'imbecility from deprivation' occurring in such cases, to consider whether the majority of them did not find their way into institutions for the mentally afflicted—and possibly do still—before accepting the apparent rarity of the condition in the literature as a guide to the frequency of the condition among

the general population. It is necessary, also, to reconsider the significance of the cases of 'imbecility' and 'insanity' mentioned in the family histories of such patients in the light of this possibility.

(3) From 1889 onwards many of the cases which we have been able to recognise as examples of congenital word-deafness were described under the name 'idioglossia,' and this was the case even as late as 1908, when Thomas described congenital word-deafness and idioglossia as two distinct conditions, believing that the latter was due to "defective development of the motor speech centre." As McCready and other have shown, idioglossia is probably the speech complication of congenital word-deafness and many cases have probably been diagnosed as such and their auditory defect not recognised. Some cases published under the name 'idioglossia' have been collected and included as cases of congenital word-deafness but these probably represent only a fraction of the total number recognised.

(4) Psychological problems and behaviour defects, as will be shown, sometimes arise as the result of the defect under discussion. In the cases of this category investigated the true cause of their condition is often unrecognised. Brônner[21], for example, in discussing certain behaviour defects, quoted two cases which fulfilled all the conditions for a diagnosis of congenital word-deafness.

(5) Quite apart from the fact that many cases of congenital word-deafness may have been missed because they were incompletely diagnosed under one of the groups mentioned above, the condition still remains much more uncommon than congenital word-blindness. For this Wallin[34] has suggested the interesting explanation that spoken language is phylogenetically a much older form of communication than written language, and therefore much less likely to be affected by biological defects or degenerations.

SEX DISTRIBUTION.

Congenital word-deafness like congenital word-blindness is apparently much more common in males than in females. Of the 50 cases collected by us there are references to the sex of the patient in only 36 cases (including our own case). Of these, 30 were males and six females, a proportion of five cases in males to one case in a female. This greater frequency of the defect in males was referred to by Coen, who pointed out that his condition 'Hörstummheit' occurred chiefly in boys. No other writer appears to have stressed this peculiar sex distribution. A similar distribution has, however, been noted in congenital word-blindness. McCready pointed out that that condition occurred more frequently in males, and found that of the 41 cases he collected in 1909, 34 were males and seven were females, a relative frequency which is almost exactly the same as that of the cases of congenital word-deafness we have collected.

THE FAMILIAL FACTOR.

A familial influence has been noted particularly in connection with congenital word-blindness, but hitherto it has not been emphasized particularly as a factor in the etiology of congenital word-deafness. Since the two defects are somewhat analogous and any discussion of the causation of the one must necessarily include consideration of influences common to both, it is necessary first to refer to the familial factor in congenital word-blindness. McCready[24] described four cases in two generations, in a father and his three sons; and in connection with a second case mentioned that the father and a paternal uncle could not read until they were ten and that their father and paternal uncle both had difficulty in learning to read until late childhood. The mother of one of Thomas's[25] cases had never been able to learn to read, and five other children in the same family suffered from the same defect as the patient. A sister of another of Thomas's cases could not read even after she had left a special school. The maternal uncle of Fisher's[19] case did not read until he was ten; Hinshelwood[50] described cases in four brothers; while both Stephenson[51] and Plate[52] described cases occurring in three generations. In Stephenson's group of cases the defect was apparently transmitted from the first to the second generation through an affected female, and from the second to the third generation through an unaffected female.

The familial factor is quite as apparent in connection with cases of congenital word-deafness, and was present in eighteen of the cases quoted and also in our own case. Hun's[4] two cases occurred in a girl and in her younger brother, while Hall's[2] two cases occurred in twins. Hale White and Golding Bird's[13] two cases were in brothers; their father spoke badly until he was twenty-three; their mother died insane and their paternal uncle spoke indistinctly until adult life. A maternal cousin of Taylor's[26] case was an 'idiot.' Guthrie's[15] second patient also suffered from pseudohypertrophic muscular dystrophy; his brother suffered from the same speech defect until shortly before his death, and a sister then aged five spoke more indistinctly than her brother. The father of one of Hadden's[10] patients died insane; two of the paternal uncles of another died insane: and in connection with a third case there was no history of insanity, stammering or defects of speech in the parents or other six children. There was, however, a paternal uncle who did not speak until he was seven and a maternal cousin who stammered slightly. Syme's[18] case was that of the third child in a family of four: the others were healthy and could hear and speak normally; but the mother was left-handed and "spoke very little." One of Thomas's[25] patients had a brother who did not speak until he was five, a sister aged five who had never been able to speak, and a paternal uncle who did not speak until he was five. The father in Tait's[28] case had a slight impediment in speech, saying "mouf" for "mouth," and his brother was an epileptic. Levy[30] discussed the familial factor in more detail than most other authors and pointed out that psychological troubles

and alcoholism were frequent in families suffering from 'alalie idiopathique de Coen,' as were also speech defects of a similar or different type. Burr, McCall, Town, Brônner, Colman, Kerr, Sainsbury, Variot, Yearsley, Wilbur, Coen and Wallin made no reference to a familial factor in their cases. In our own case a maternal aunt spoke first at the age of four and did not speak at all clearly until the age of ten, another aunt was reputed to suffer from "nerve-deafness" and an uncle on the mother's side was backward in speaking and at three could not pronounce words well enough to be understood.

Thus it is evident that there is a definite familial factor in cases of congenital word-deafness but no definite evidence to show in what manner the defect is transmitted from one generation to another. The presence of this familial factor is a matter of some importance when one considers the causation of the two defects and particularly of congenital word-deafness; and seems to support the presence of a biological defect rather than the results of birth injury as the essential cause of the condition.

PATHOGENESIS.

In the absence of autopsies on patients who have suffered from congenital auditory imperception, one can only speculate upon the pathology of the condition. Even if autopsies were available, it is probable that they would reveal little or no direct evidence of focal lesions in the brain to account for the clinical symptoms. In the case of similar conditions affecting the cells of the pre-Rolandic motor cortex and other parts of the brain, a careful microscopical examination has often been necessary to elucidate the anatomical changes associated with a clinical picture which appeared to be due to a lesion in that part. Consequently, we can only consider probable causes and discuss the evidence for and against them.

The probable causes of congenital auditory imperception are (1) trauma, and (2) a biological variation of the nature of aplasia affecting both hemispheres of the brain behind the temporal lobes.

In favour of trauma as a cause there is no evidence apart from the occasional report of a difficult labour during which the patient might have been exposed to injury. Against it is the need for both sides of the brain to be affected by the injury before the condition can be produced. In the event of one side being injured, the opposite side, as a rule, takes over the functions in relation to speech for which it is responsible. If due to trauma, it might be suggested that the comparative rarity of bilateral injury would account for the rarity of congenital auditory imperception.

In support of a biological variation as the cause are the following points: (1) There is a definite familial incidence in one-third of the cases reported in the literature. (2) In order to produce the clinical picture, the lesion must be a bilateral one, and most conditions believed to be due to local aplasia of the cerebral cortex are bilateral in distribution. (3) With education, a certain

degree of recovery results, a feature which has been noted in cases of aplasia affecting the motor cortex and the cerebellum. On the whole, the balance of evidence appears to be in favour of aplasia of the post-temporal cerebral cortex on both sides as the essential pathological condition underlying congenital auditory imperception. It is true that cerebral aplasia affecting the motor cortex and cerebellum rarely shows a familial incidence and on that account it might be preferred to correlate the pathological condition with the abiotrophies, which include the heredo-familial ataxias, rather than with the congenital aplasias. Such a consideration does not, however, invalidate the evidence in support of a biological variation as the essential cause of the condition.

CLINICAL FEATURES.

The clinical features of congenital word-deafness are many and varied. The general clinical picture presented by patients suffering from the defect may be appreciated from the following translation of Lévy's definition of the abnormality (as already mentioned). "This type of mutism is recognised by the following positive and negative characteristics—(1) children who are normal physically and mentally; (2) hearing normal; (3) the peripheral organs of speech properly formed and functioning normally; (4) absence of paralysis, atrophy or contracture of the body or limbs. Usually they are children from four to ten years of age, whose physique and intellect appear to be normal, who in a word have all the appearance of children of their age except that they do not speak at all or have a rudimentary language for their age." Though this definition draws attention to many of the more important manifestations and is sufficiently complete to serve as a starting-point for a discussion of the clinical features of the condition, it is wanting in several important particulars and needs to be elaborated considerably.

(1) *The defect is present from birth.* It is rarely observed, however, until the child is old enough for the parents to notice that his acquisition of speech and his understanding of speech and language are defective. Hence in the cases reported in the literature there is rarely any reference to the condition of the child during his first three years of life beyond the statement that he had never been able to learn to speak. We were fortunate enough, however, to obtain a history which emphasized several unusual features of our patient during infancy. In spite of the fact that he was a normal child in all other respects, he had a peculiar cry—low and moaning—quite unlike that of other children of his age. The monotony of the cry and its lack of variation with different affective states were particularly observed by those in attendance upon him. Though he took notice of things about him in the usual way and at the usual time, he did not do so when spoken to. He did not make the responses usual in an infant of his age to those who spoke to him, and in fact pushed them out of the way so that he could see what was going on about him. A child the

subject of this defect does not attempt to say words at the usual time. Although those about him attempt to teach him to speak by getting him to repeat sounds and words after them, they find that he never attempts to say words of his own accord. They notice that, when he must produce some sound to attract the attention of those near him, he usually makes a sound or noise which bears no resemblance to speech. After he has reached the age of eighteen months or two years they complain that he does not learn to talk as other children of his age.

(2) *Hearing is normal.* This is often appreciated by the parents of the child long before he is brought for advice. It is also appreciated by teachers of the deaf who recognise that there are sometimes children in classes for deaf-mutes who can hear and yet cannot speak. This is an aspect of the condition which has been stressed not only by Levy but also by other writers who have reported cases. In fact, it is often thought by those who are associated with the child that he must understand language as it is obvious that he can hear; and this error is not absent from the descriptions of the earlier cases reported in the literature. One of Wilbur's patients was said to "understand simple language spoken to him or in his hearing"; another could hear perfectly well; a third was said to understand ordinary commands; while the fourth knew his name and understood some simple language addressed to him. One of Burr's patients was said to understand all that was said to him; another was thought to understand spoken language as well as an average child of his age; while a third heard perfectly well as was shown by the fact that he repeated what was said in his hearing. McCall's patient took no notice when spoken to and might easily have been mistaken for a deaf child, but a noise attracted his attention. His teachers were sure he was not deaf. Tait's patient could hear; both Thomas's patients could hear perfectly well; Kerr's had good hearing; and Sainsbury's showed no deafness. In our case the auditory apparatus was normal in every respect. In many of the cases reported as idioglossia or a similar speech defect, no direct reference was made to the hearing beyond the statement that, apart from speech, the patient was normal in every way. Guthrie was of the opinion that in some of the cases referred to by him, the hearing was anything but acute as far as mere audition of sounds was concerned. Some writers, including Spencer Watson and Langdon Brown, believed that the speech defect was due to a defect of hearing short of actual deafness, and others, including Levy, believed that impairment of hearing as the result of nasopharyngeal growths and adenoids was sometimes associated with the defect. As Guthrie pointed out, however, the effect of these conditions in producing defective utterance would not alone account for the abnormality of speech described as idioglossia. In some cases, if care is not taken to test the hearing systematically, the impression may be produced that the patient is partially deaf owing to the fact that he cannot distinguish variations in sounds. This source of error was referred to by McCall, and may

have been responsible for the opinions expressed by Spencer Watson, Langdon Brown and others, that adenoids and defect of hearing short of deafness were responsible for the speech defect associated with congenital word-deafness. In addition to the four cases quoted from Wilbur's series, there were two other patients in the same series who were described as partially or slightly deaf, but otherwise showed a clinical picture very suggestive of congenital word-deafness. It is possible that these two cases were actually examples of the condition and that the patients were thought to be partially deaf because they had difficulty in appreciating variations in sounds. Hence it is necessary to emphasise the need for a careful and detailed examination of the hearing before arriving at a definite conclusion concerning a suspected case, for not only may patients suffering from the defect be thought to be partially deaf when they are not, but those suffering from a minor degree of deafness, such as in the case reported by Drinkwater, may have their deafness unrecognised and be diagnosed in error as cases of congenital word-deafness.

(3) *Spoken language is not understood when spoken in the patient's hearing and out of his sight.* This is the essential feature of the condition, and the basis of the varied symptomatology which has grown up around it. Two precautions are necessary in testing this aspect. In the first place, it is essential to examine the patient separately for the comprehension of spoken language which he *hears* and for that which he *sees*, especially when the patient has, as many of them have, been in a school for the deaf and has been taught lip-reading. Neglect of this precaution has probably been responsible for the varied descriptions in the literature of the patients' ability to understand spoken language and for the fact that in many cases the true nature of the defect has not been recognised. Even in a patient who has been taught lip-reading a careful examination in two stages as indicated above will reveal a very obvious difference between the ability to comprehend spoken language 'heard' and that 'seen.' In many of the cases included in our series on other evidence this precaution was apparently not observed. For example, one of Burr's patients was recorded as being able to understand all that was said to him and another was thought to understand spoken language. The second precaution is even more important. Tests which depend upon the patient's repeating what he hears should not be employed, for, as will be shown later, these patients can often repeat parrotwise the sounds they hear though they do not understand their meaning. Only those tests which demand that the patient should understand the meaning of the sounds and words he hears should be employed. The descriptions of many of the earlier cases investigated were also deficient in this respect. Guthrie, for example, wrote of his first case as follows: "Whilst her attention was engaged she could repeat most words correctly at dictation, but she soon looked vacantly round the room and her speech became gibberish although she still continued mechanically to utter words after me." Frequently, it is only when care is taken to eliminate the two sources of error

mentioned that the true nature of the defect is appreciated. As Wallin pointed out, spoken language is heard but not comprehended. "The difficulty is not in the auditory perception of the spoken sounds, but in the recognition of the acoustic word imagery. Speech sounds to the word-deaf like a foreign language, a meaningless jargon of sounds." In his case, the patient, although aged seven and a half, was rated only 2–4 years in the Binet-Simon test, in which success is intimately dependent upon the understanding of spoken instructions. In Syme's case it was recorded that "he has no idea of the meaning of sounds. A book is touched and the word 'book' is spoken to him and repeated in a manner by him several times. If now the book is pointed to and he is asked by speech and signs what it is, he never attempts to say 'book.'" Thomas described a patient who heard a watch at a distance from either ear, and heard words readily but understood none. His second patient heard a bell, a whistle and all musical notes readily and responded by raising his hand: he made an attempt to repeat words heard but could attach no meaning to them. Town's patient did not understand the words he heard "as was shown by his failure to comply with requests or point out objects."

(4) *In many cases the patient can repeat sounds and words he hears, but he cannot repeat a long series of sounds or words.* This feature has been demonstrated in a previous communication[2] in the results of those tests which depend upon the repetition of a phrase of five words. This ability to repeat sounds heard varies considerably. In some cases the imitation is almost perfect, in others only the fact that the same number of syllables is repeated can be appreciated by the observer, and in some there is no attempt at all at repetition. This feature of the condition is often apparent at once when the patient is asked a question, for he may repeat it parrotwise but make no attempt to supply an answer. Syme's patient attempted to repeat the words said in his hearing when the eyes were covered—he was right as regards the number of syllables but not in the words themselves. In one of Wilbur's cases it was found that, though his mother said he could speak, he repeated what he heard or the question but made no attempt at spontaneous speech. Yearsley's patient simply repeated the speech she heard in the following manner:

"*Q.*—What is the time? (By ear alone.)

A.—What is the time? (Mumbled without intelligence.)"

One of Thomas's patients repeated the command "Get me a pencil" as "Det me a pentil" but made no attempt to carry out the command, and his second patient made an attempt to repeat the words he heard but could attach no meaning to them. One of Burr's patients could repeat words immediately after he heard them, but no new words could be fixed in his memory, although his memory for events was good. Variot's patient could be persuaded to repeat a few syllables parrotwise but in doing so he spoke in a somewhat explosive manner and with the syllables spaced.

In carrying out the examination of our patient it was difficult to persuade him to obey a command in silence. For example, in performing Head's 'The naming and recognition of common objects[2]' test by getting him to point to an object named verbally by the observer out of his sight, in most of the tests he repeated the name of the object imperfectly after the observer. The inability to repeat correctly more than one or two syllables was shown most clearly in 'The man, the cat and the dog' test. In the first test, when asked to repeat the words "The cat and the dog" after the observer he repeated them more or less successfully as "Ge kaht ahn ge doee," but with later tests he was unable to repeat more than the first word in his own way. For "The dog and the cat" he repeated "Ge dog ge ahn aich" in which only the first two words corresponded to the original. Finally, after a series of tests, the words repeated bore no resemblance to the original words and, though the fact that the phrase consisted of five words was still remembered, there appeared to be complete confusion concerning the relative position of the words. In the last test he repeated "The man and the cat" as "Ge doee doee ge taht." It seemed as if in the earlier tests the words of the phrase were remembered for a short period, but after that only the fact that there were five words in the phrase was appreciated.

This ability to repeat a limited series of words after the observer is liable to cause some confusion in the examination of such patients. Our patient was reported to have been able to say his name and "Cockadoodle-doo" at the age of three years, but on closer investigation of the circumstances it was found that on each occasion he simply repeated the word after someone who was trying to teach him. In all the tests applied to him depending upon the repetition of sounds heard there was an obvious element of perseveration. In 'The man, the cat and the dog' test, his version of the word 'dog' persisted through all the repetitions after the first one; while, on asking him questions either by ear alone or by lip-reading, his reply was often his name whatever the subject of the question.

It is apparent therefore that such patients often have a limited ability to repeat the sounds they hear in an imperfect manner, and sometimes reply with a stock word or phrase to any questions which may be put to them. It is equally obvious that any method of examination which does not depend primarily upon the appreciation of the words heard may not reveal the true nature of the defect from which the patient is suffering.

(5) *Sometimes the patient fails to understand and appreciate musical sounds, and sometimes even cruder sounds. The ability to recognise musical sounds and more particularly crude sounds may, however, be perfectly normal.* Our patient could not recognise discords produced by himself on the piano when his eyes were covered, and failed to appreciate the difference between an unmusical noise and the sound of a tuning-fork. In the latter test he indicated in each case by signs that it was a tuning-fork he heard. When asked to repeat a

series of notes sung in his hearing, he changed the inflection of his voice from one note to another but failed to reproduce the notes correctly. Some crude sounds he was able to recognise at times, for the noise made by the wind sometimes made him afraid. He was also able to associate the sounds of the footsteps on a pathway or the noise produced by opening a door with the arrival of his father. His ability to recognise crude sounds apparently varied from time to time: and an examination of the cases reported in the literature leads us to believe that the recognition of crude sounds and of musical sounds varies considerably in cases of congenital word-deafness, the variation probably depending in some degree upon the severity of the defect and upon such a general factor as fatigue. In one of Guthrie's[15] patients the 'musical sense' was singularly wanting, and he apparently rendered pieces by rhythm rather than by melody. The same method was apparent in the attempts made by our patient to play short pieces on the piano. One of Thomas's[25] patients could hear a bell, a whistle and all musical sounds with ease and responded by raising his hand, but it is not at all clear whether he appreciated the differences in the various sounds. Syme's[18] patient was fond of music, heard at once if a street organ began to play nearby and could hum part of a tune. Our patient was fond of listening to organ music, but sometimes he would listen to it for only a few minutes and then his attention would wander.

(6) *When lip-reading has been learned, as it has been by many who suffer from the defect, the patient may be able to understand language easily by sight.* Although our patient had had only a limited education in lip-reading, it was obvious in the tests applied to him that he could understand a fair proportion of the speech which he saw, which was in decided contrast with his entire lack of ability to understand speech which he heard. Such words as 'match,' 'card,' 'florin' and 'penny' were repeated imperfectly by the patient when spoken by the observer where he could see, and the command to point out the object was obeyed correctly. In the earlier cases recorded this ability of the patient to understand language by sight frequently produced the impression that the patient could understand spoken language.

(7) *Speech is absent or exceedingly defective.* If the patient learns to speak at all, speech is acquired late and often not before five to twelve years of age. Speech is rarely used spontaneously, and, if it is, phrases of only two or three syllables are employed: it lacks stress and intonation. When communication by signs or pantomime fails, speech is sometimes fluent and of the nature of jargon—a series of unrelated sounds without grammatical structure or sequence: and it cannot be understood by those who are unaccustomed to the patient. Even after attempts have been made at special education in articulation speech may be permanently defective, or it may be of the nature of an individual language based upon the misunderstanding and mispronunciation of the sounds and words heard, one in which special sounds and groups of sounds are used constantly to indicate certain objects and affective states. The

characteristics of the speech defect and its relationship to the original defect are discussed in detail under a separate heading.

(8) *There is some difficulty in appreciating the meaning of written and printed symbols*, even when the patient has been brought up in a good environment and has received such education, apart from special education, as it is possible for him to receive. In many cases, of course, education has not proceeded sufficiently for the observer to discover whether this defect is present or not. In tests depending upon pointing to objects named in print, our patient was able to obey the command correctly in the majority of tests, but in a number of them he failed to appreciate the meaning of the printed symbols and carried out the command incorrectly. This defect was found to be a handicap in his school work, for when given directions in writing or print he would often simply copy the writing or print and fail to obey the directions contained therein. This defect in the visual field renders the education of patients suffering from congenital word-deafness much more difficult than it would appear to be at first sight. The ability to appreciate and remember pictures and the qualities of form and shape is as a rule prefectly normal. Games can be indulged in with zest and mechanical toys and machinery arouse the full interest of the patient. The ability to appreciate arithmetical symbols is as a rule fairly good, but at times there is some inability to understand the significance and details of the arithmetical processes involved in simple exercises and certain bizarre effects are produced as a result. These were illustrated fully in a previous communication. As will be shown later, this defective appreciation of the meaning of written and printed symbols is merely a part of the 'aphasia' which complicates the original defect.

(9) *Attempts at writing reveal certain errors which correspond in general to the errors found in oral speech in those patients who have received some education.* These again are evidences of the 'aphasia' which complicates the original defect. In the absence of detailed methods of investigation this feature of the condition has not been referred to in many of the cases reported in the literature. One of Brônner's patients (already alluded to) wrote " I never went any place outside of Chicago " as " I necar wast ene plaes atsed in Chicago." Attempts at spontaneous writing, when they can be made by the patient, often produce ludicrous results. Spelling is defective, and even simple words are misspelled. It is noticeable, however, that words to which the patient has become thoroughly accustomed, such as the name of the town in which he lives and his own name, are rarely misspelled. As far as could be gathered from the investigation of our patient, though the spelling of the names of objects pointed to was apparently quite incorrect, certain groupings of symbols were used more or less constantly for the same objects. In this way the language expressed in writing corresponded with that expressed in oral speech. By our patient a match was constantly named 'mat,' but was expressed in writing at different times as MTLLA and MTLLD. 'Hand' was written at

different times as HTND, HLMD, HLED and HSND. This defect in writing or spelling may be noticed by the patient's teacher as a constant tendency to make such mistakes as GRIL for GIRL. Although rather overshadowed by the more serious symptoms of the condition and thus of little importance in diagnosis, this inability to spell even the simplest of words correctly may be found to be a serious handicap when attempts are made to educate the patient through channels other than the auditory.

(10) *Writing from dictation is impossible when the speaker is out of the patient's sight*, but may be possible to a limited extent when the patient can see the speaker and has become fairly proficient in lip-reading. Here again, however, if each word is not dictated separately, the same errors of spelling appear as in spontaneous writing. Copying can be performed accurately and, in fact, is often done with much greater attention to detail than is shown by a normal child of the same age.

The patient can express himself in pantomime and by signs, and appreciates the use of pantomime and signs by others. As a rule, physically, he is perfectly normal. There are no defects in the organs of articulation, and physical examination of the nervous system reveals no abnormalities whatever. Some writers have suggested that other congenital defects are sometimes associated with congenital word-deafness, but an examination of the case-reports which have appeared in the literature has revealed only one case in which another congenital defect was present.

(11) *The mental condition* of children who are the subjects of congenital word-deafness, and the special psychological and educational problems associated with the defect, are considered in detail at a later stage. The child is backward at school, if indeed he has ever reached a stage which would justify sending him to an ordinary school. He is unable to obtain information as quickly and as easily as other children of his age, mainly because the channels of communication are limited. Because of his difficulty in understanding speech and his lack of spontaneous speech he is often thought to be mentally deficient, but, as a rule, his mental condition is normal if he has been brought up in a good environment and treated by his parents and teachers with sympathetic understanding. If neglected, he may perhaps become an 'imbecile from deprivation' in the sense that his lack of speech and difficulty in obtaining information have rendered him incapable of competing economically with his fellows. If not handled with understanding, he may become mentally isolated and develop abnormal psychological reactions to his defect, to his environment and to society. Such abnormal psychological reactions may be aggravated if he is eventually placed in some occupation which is unsuitable because of his personal handicap. His behaviour may then be in some degree antisocial, and may lead him into the courts for offences against the person or society. Because of the much more severe economic handicap entailed in the absence of speech and the lack of understanding of spoken language, such evidences of

delinquency are even more likely to occur in sufferers from congenital word-deafness than in those who suffer from the corresponding visual defect. A correct diagnosis of the patient's defect, special education adapted to his particular needs and the choice of an occupation which calls into play his special abilities and places as little stress as possible upon his personal defect, produce in the majority of cases a normal adjustment to society and ensure the normal mental development of the individual.

THE SPEECH DEFECT.

Since the evolution of speech depends upon the integrity of auditory perception the subjects of congenital word-deafness have difficulty in learning to speak. Other causes of delay in acquiring speech are a physiological slow rate of development and deafness resulting from a peripheral lesion; these must be excluded before the condition can be diagnosed definitely as congenital word-deafness. According to McCready, children with such a speech defect are of three types—(1) those in whom all inclination to speech is lacking; (2) those who are dumb without being deaf for ordinary sounds; and (3) those who sometimes enunciate a few definite sounds which they use in connection with definite objects and affective states. Those of the first and second type are fairly common; and in fact all the children suffering from congenital word-deafness might be included in one of these types before they have acquired the few words or sounds which serve them as speech. A few of them acquire some form of speech by the age of three years; more often they have acquired no such means of communicating with their fellows by the age of five years; and cases are on record in which speech has not been acquired before twelve years of age. One of Wilbur's patients did not attempt to speak until he was nine, and at thirteen could say only a few words which his father alone could understand. Another patient had made no attempt to speak by the time he was eight; while a third began to use only a few single words uttered rapidly at the age of twelve years. One of Hadden's patients was four before he could make a few sounds; Syme's patient had not begun to talk or to make use of the usual childish expressions at eighteen months; Kerr's patient did not speak at all until he was taught articulation; and one of Thomas's patients did not speak until he was seven. Tait's patient could not utter a single word at the age of five; some of Levy's patients did not speak at all; one of Burr's had never made an effort to talk at three, another was unable to speak at twelve, and a third could say only 'yes' and 'no' at seventeen. Such children as these, for all practical purposes, are dumb and are able to communicate with their fellows only by signs and pantomime. They may find their way into schools for the deaf but sooner or later they are found to be able to hear and yet lack the power of spontaneous speech. They are often misunderstood and are thought to be mentally defective or merely lazy and inattentive.

It is more common, however, for children suffering from congenital word-deafness to acquire a few sounds and words with which they are able to make

their wants known to those in constant association with them. Their success in expressing themselves in this way varies considerably. Some can make only a few sounds, while others have a fairly large vocabulary which includes sounds and expressions for most of the objects and affective states which come within their experience. In the earlier cases reported, the details of this method of expression were not referred to with any minuteness. One of Wilbur's patients uttered sounds which only his father could understand, and even after a long period of education in articulation produced his words deliberately and imperfectly. Another expressed himself chiefly by pantomime and at the age of twelve supplemented this method of communication by a few single words imperfectly pronounced. Sainsbury described the speech defect in detail and mentioned that the sounds used by the patients were not as a rule intelligible to strangers. In naming the alphabet, his patient could name correctly only the letters C, P and T; and rendered B and D as P and T. For the remaining letters of the alphabet there was apparently no relation between the normal and the abnormal sounds, the pronunciation of Q being an exception. There was a frequent repetition of the S sound in the naming of the letters. One of Hadden's patients could pronounce all the vowel sounds, and in repeating the letters of the alphabet mispronounced the names of the letters C, H, I, Q, R, S and Z. Many other defects appeared when he attempted to combine sounds into words, and it was noted that he was slovenly in ending words. He repeated the Lord's Prayer in the following way: "Ane Fave, which art in en-en, Annow be ma name, Ney in-dom um, Vy will be done in ear va it e in ennung, E uh vith day a dai-y be, A fogee uh for eheh-e-ay, A we foh-eev vem vat eheh-futh a ain uh, And ee uh not va tentay-ung, Buh deuce uh fon evil, Foh ine in e ingdom, E power and i o-wy, Foh ee an ee, Amen." Hale-White and Golding-Bird termed the defective condition of speech 'idioglossia' because it gave the impression of an individual language. One of Guthrie's patients was described as talking gibberish when her attention wandered: and the vocabulary of his second case consisted almost entirely of the consonant sounds B, P, D, T, K and of various vowel sounds. One of Colman's patients pronounced the vowels correctly except 'o' in 'not' which he pronounced as "naht." His version of the Lord's prayer (already quoted) was as follows: "Oue tabde ne nah e nedde, anne de di na: i tede ta, i du de di on eeth a te e edde. Te ut te da oue dade de, e didde ap tetedde, a ne adin to tetedde adase us, ne notte totate, mime, utte enu, to i aitevene, pore e dande, to edde a edde. Ame." He sang fairly well but his singing showed the same defects in articulation. After six weeks' training he improved very much and rapidly picked up the sounds for the clear pronunciation of the different letters and words except K and G. A second patient could pronounce the vowels well, but used T, D and N for most of the consonants; he could not pronounce the letters F, V, TH, SH, R, K, and G. His speech was quite unintelligible to those unaccustomed to him. Syme's patient had never spoken in an intelligible

manner at the age of eight, though he occasionally used non-intelligible sounds such as "ow" and "bow" especially when he was angry. Yearsley's patient could pronounce no words properly even after three years at a deaf and dumb school. Her spontaneous speech was slipshod and indistinct, and showed a tendency to replace SH by S; D, G, Z, and T often being mistaken as well. When asked a question she mumbled the question itself without intelligence. Burr described the speech of one of his patients as simply a series of "inarticulate sounds." McCall's patient had "markedly defective speech" and its intonation resembled in a slight degree that peculiar to cleft palate. Tait's patient phonated a sound like 'ta' or 'ah' suggestive of cleft palate and made these sounds do duty for everything. One of Brônner's patients showed numerous verbal inaccuracies in his speech and "the English he employed was exceedingly poor." His enunciation was exceedingly faulty though his vocabulary showed a fairly wide range. Certain syllables were slurred over. Wallin described the speech defect associated with congenital word-deafness sa follows: "He expresses himself through speech with difficulty. His speech may even be reduced to a jargon of gibberish or reiterated words or letters without grammatical structure."

The speech of our patient has been described in detail in the previous communications referred to. Its general characteristics were as follows: (1) it sounded like gibberish when one was unaccustomed to him and seemed to have no semblance of grammatical formation; (2) speech was rarely used spontaneously and on the rare occasions on which it was, it was confined to single sounds or words; (3) single words or sounds were as a rule used alone in reply to questions, but when these or communication by signs were thought to have failed, there was produced a succession of sounds which seemed to be jargon and not true speech; (4) upon closer observation, certain sounds or series of sounds were found to be used constantly for the same objects or affective states; (5) words such as those for the days of the week were begun with a sound which in some cases resembled the correct one and then trailed off into a long or short vowel sound; (6) there was an element of perseveration in producing a number of words usually pronounced in series, e.g., in counting, the sound "iki" was used to end the name of most of the numbers after sixteen; (7) individual expressions were used constantly for definite objects, e.g., "pickapek" for "pocket"; (8) the speech was monotonous, toneless, indistinct and lacked stress and obvious division into syllables; (9) upon continued observation, the patient's method of expression could be learned and his meaning more easily understood, as in learning a foreign language by associating with one who spoke it; (10) the speech could be readily understood by those in close contact with the patient and by other children of the same age.

Since the speech defect is the most obvious symptom of congenital word-deafness and the one which most often draws attention to the condition, it is necessary to discuss in detail the nature of the defect and its relation to the

original condition. For long it was regarded as an independent condition and its relationship with the defect in the auditory field was unsuspected. In the earlier cases described, the speech defect was thought to be the result of imbecility, idiocy or defective mental development. Wilbur described his cases illustrating the defect as examples of 'aphasia' supervening in mentally defective children, but made it perfectly clear that they belonged to a class of their own—one including mentally defective children who could repeat words after the observer but could not originate them. Arguing from the mental deficiency which he believed was present in all his cases, he stated that in the majority the absence or defect of speech was due to "lack of volition" on the part of the patient. That his argument was fundamentally wrong is shown by the fact that the descriptions of his cases contained references to no defects, other than those of speech and 'hearing,' which might suggest the presence of mental deficiency. This aspect of the condition was discussed as late as 1912 by Burr who showed that the speech defect in such cases was in no way dependent upon mental deficiency but that such children might become 'imbeciles from deprivation' just as deaf-mutes did formerly. A cursory examination of the patient might have suggested that the speech defect was the result of mental deficiency rather than that the mental deficiency and the speech defect were due to a common cause—congenital word-deafness.

Later it was suggested that the speech defect was a species of 'atavism' or 'a sport in language,' the suggestion being that such children used words of their own invention and in doing so harked back to the speech of their ancestors. Hall's two twins of German extraction were said to speak a language of their own thought to resemble German, while the speech of Hun's two patients was thought to resemble French. Crane reported instances of what he believed to be "atavism in speech," but it was noticed by Guthrie that the terms employed by his patients were simply misapplied and mispronounced words which the children had actually heard and had understood only imperfectly. Romanes[53] pointed out that the examples quoted were not striking and that the apparent resemblance to the language of their forebears could be easily explained otherwise. As Guthrie pointed out, the strongest argument against the spontaneous evolution of speech in such cases was the fact that deaf-mutes never talked at all unless they were taught to do so.

In 1891, Hale White and Golding Bird introduced the term 'idioglossia' to describe the defective speech, because it gave the impression that the children were speaking a purely individual language. Though Guthrie defended the use of the term on the grounds that the language used produced the same effect as if a new language were heard, he made it perfectly clear that the defective speech was nothing more than imperfectly pronounced English due to defective audition in the sense that the distinctions between sounds were not understood or not heard. Thus he recognised some connection between the speech defect and a defect of auditory perception, but appeared to think that it was simply an

exaggeration of defective modes of speech which were fairly common in minor degrees without being regarded as in any way morbid. The same attitude was adopted by Wyllie who grouped the defect among all classes of defective utterances and thought that it was due in some measure at least to imbecility. As late as 1908, Thomas regarded the defect in the same light, included it with other types of defective speech and believed that they were all due to defective organisation of the motor centre for speech.

Sainsbury had, however, in 1889 suggested a relationship between a speech defect which bore all the characteristics of idioglossia and a congenital defect in the auditory field analogous with that in the visual field known as congenital word-blindness. Though Kerr had in 1900 described a case of congenital word-deafness in detail and pointed out that children suffering from the defect did not talk properly until taught articulation, the separation of idioglossia and congenital word-deafness into two distinct entities persisted until 1910 when McCready suggested that idioglossia was due to partial word-deafness. In his opinion the speech defect was due to the child repeating sounds as he heard them. This view of the defect served the purpose of drawing attention to the speech defect as the important complication of congenital word-deafness, but failed to account for all the features of the defective speech.

As a result of Marie's conception of speech as a function which acted as a whole and in which each part was dependent upon the others, and to Head's detailed method of investigating aphasia and his revival of Hughlings Jackson's view that the function of speech was on a much higher level than it had been regarded for the previous fifty years, it became apparent that the relationship of the speech defect to congenital word-deafness could not be explained fully in the simple manner suggested by McCready. Head regarded the disturbance in the auditory field as belonging to a lower order of functional disintegration than aphasia and grouped it under the heading of auditory imperception. He pointed out that in addition to the difficulty in appreciating the meaning of words the patient had at some time or other difficulty in recognising the meaning of crude sounds. Wallin also referred to the fact that in addition to inability to recognise the meaning of words, the patient sometimes suffered not only from amusia, or the loss of the ability to appreciate melodies or to recognize musical sounds, but also from what he termed "acoustic aphasia" or inability to recognize sounds and noises. Referring to the latter defect, he said that "it is invariably accompanied by word-deafness, and, in the case of musical persons, by amusia, and frequently by paraphasia, so that their speech resembles that of confused lunatics."

The investigation of a patient by Head's methods shows also that the speech defect is due to something more than the child's repeating words as he hears them. It is true that when asked to repeat sounds and words after the observer, he does so in a manner approximating to the original but differing from it in

the monotony of tone and the lack of stress, but this method of repetition parrotwise after the observer draws attention to a very minor part of the speech defect and fails to reveal the errors which are evident in spontaneous speech. The results of the examination of our patient by Head's method have been given in detail elsewhere. From them it is evident at once that in such a case there is present a much more widespread defect than can be explained by congenital word-deafness alone and a complicating speech defect which has arisen as a result of the child's repeating sounds as he heard them. The defect in speech is obviously due to something more than the mispronunciation of imperfectly heard and misunderstood words; defects in a similar form appear in the expression of speech in writing, and in addition to the original defect in the auditory field there is an obvious though less apparent defect in the understanding of written and printed language. Consideration of the detailed results of this examination shows that:—(1) there is complete absence of auditory perception for words, musical sounds and certain crude sounds, though at times such crude sounds as those made by the wind are understood and appreciated; (2) there is present a type of 'aphasia' involving chiefly the oral expression of language, and to a lesser extent the expression of language in writing and the visual appreciation of written and printed characters—in short, a type which is the same as Head's 'verbal aphasia.' Thus it would appear that the fundamental defect—congenital word-deafness—might be more correctly described as a defect in auditory perception, and that the clinical picture of the condition as revealed in oral, spontaneous speech depends upon a complicating 'verbal aphasia,' the defective speech associated with which is the same as the 'individual language,' 'idioglossia' and 'defect of articulation' of various writers. The explanations of the nature of the speech defect given in the literature, in which it is variously described as the result of imbecility, due to lack of volition, 'a sport in language,' an individual language or simply mispronounced English due to the child repeating words as he hears them, are obviously at fault. In support of all the views but the last there is no satisfactory evidence at all, and against the last view is the fact that there is some defect in all aspects of speech and not in the oral expression of language alone.

There remains one point to be discussed—whether the term 'aphasia' correctly describes the condition which is present. In its strict sense the term means the loss or absence of speech which has already been present, and is used to describe the disturbance of speech in those in whom the function has previously been perfect. It has been suggested by Tait, Burr and others that the term is not strictly applicable to those cases which depend on a congenital defect. In default of a better term, however, the term 'aphasia' will no doubt continue to be employed since it has already been applied for many years to the condition under discussion. It should, however, be understood that it is used in a sense somewhat different from that in which it is employed in adult cases.

It is apparent, therefore, that although there is a certain speech defect due to the mispronunciation of words heard when the patient repeats them after the observer, the chief speech defect is due to a 'verbal aphasia' which complicates the original auditory defect. When, however, the patient has learned to speak by education through the visual channel and particularly by means of lip-reading as taught to deaf mutes he pronounces his words as he sees and hears them pronounced, but as he is still unable to appreciate the differences between sounds his speech is somewhat different from that of the normal individual. Depending upon the success of his education, his speech may vary from a collection of gibberish with an occasional normal word imperfectly pronounced to words and sentences which are readily understood even by those unaccustomed to him, but is still deliberate and lacking the intonation of the speech of a normal individual.

THE MENTAL CONDITION.

Opinions as to the mental condition of patients suffering from congenital word-deafness have varied considerably; for a time they were thought to be always mentally deficient; but at present the general tendency is to regard them as at least of potentially normal mentality. All Wilbur's cases were found in an institution for idiots, but it is interesting to note that the clinical descriptions of the cases quoted by him revealed no evidences of idiocy other than the defect of speech and the difficulty in understanding speech, and no features which could not be explained as the results of these defects. Coen insisted that such patients were normal physically and mentally. Sainsbury's patient—a girl five and a half years old—was a bright-looking, good child, was useful in the house, could run errands and amused herself with her doll. Hadden did not think that any of his patients were deficient in intelligence; Taylor's patient was intelligent; while, of Hale White and Golding Bird's two one was "sharp and able" and the other was intelligent. Colman attempted to explain why such children were thought to be mentally defective. He pointed out that "although the children are often intelligent and quick, the difficulty of making themselves understood gives other people the impression that they are idiots." Kerr's patient was very intelligent, his only defects being that he could not understand words or speak. Syme's patient—a child of eight—was bright and cheerful; clean in his habits; and showed more intelligence than the average. He could dress and feed himself, played with other children, drew well and copied the names of shops he had visited. The patient described by Guthrie was quite intelligent but his education had been neglected on account of his physical infirmity. He could not, however, concentrate his attention for more than a short time. From this patient and a number of others he reviewed, Guthrie concluded that the subjects of idioglossia were not actually unintelligent but had some inherent inability to make use of the faculties of attention and observation which are necessary for the acquisition of correct speech.

The patients described by Thomas, Yearsley and Variot were apparently quite normal mentally. Town found no evidence of general mental defect in her patient, who made his wants known by gestures, understood signs, showed evidence of a retentive memory, and was very interested in finding the causes of events. Tait's patient was no different from other children except for the inability to talk. He was obedient, could run messages, played with other children, and recognised people he had seen before. McCall thought that her patient was somewhat less intelligent than a patient suffering from congenital word-blindness, but more intelligent than an ordinary mental defective. Levy followed five cases of 'alalie idiopathique de Coen' for a long period, and stated that, of these, two became normal individuals and three were still backward, one of them then aged twenty-one not having yet developed a comprehensible language or developed in accordance with her age. He enumerated the changes associated with the defect as memory defects, poor attention, and ill-developed intelligence. Levy thought that the two patients who had become normal might have been better mentally if they had not suffered from the defect, and he concluded that the existence of dumbness in children with normal intelligence remained to be proved.

In spite of the fact that many of the patients described were intelligent, the idea was fairly common until 1912 that when mutism occurred without deafness it was due to imbecility. Because of this idea some institutions for the education of mutes would not admit or even examine children who could hear and were mute, as it was thought that they were imbecile and unsuitable for such institutions. It is not always easy to determine the true mental state of such children especially when they have received little or no training. According to Burr, it is possible to judge their mental development only by their general conduct and behaviour, the kind of things they take an interest in, whether they show normal inquisitiveness, their powers of emotional inhibition and how they respond to attempts to teach them not only speech but knowledge of any kind. On this basis, Burr concluded that some mental deficiency was present in some cases of congenital word-deafness, that it was the result of the speech defect and not the cause of it; in fact, that such children became 'imbeciles from deprivation' when they were exposed to a poor environment, but were potentially normal and would become intelligent as other children if treated in the proper manner. He certainly believed that disease or lack of development of the auditory speech centre always produced some and usually marked mental deficiency, but was careful to point out that such mental weakness was due to an unavoidable accident of environment and was not a necessary result of the disease. His first patient was good-natured and intelligent considering that he had had no education. With education "his intelligence or rather his knowledge increased a great deal." He learned things quickly and well, his memory was excellent and his moral sense good. A second patient was normal mentally, and a third had fair

intelligence, was obedient and good-natured and was able to take care of himself.

Brônner discussed the defect chiefly from the point of view of the behaviour and educational problems involved. Judging by the descriptions given her patients were mentally quite normal apart from the tendency to delinquency. After long observation of his case, Wallin concluded that the patient was somewhat subnormal intellectually. Discussing the mentality of individuals suffering from congenital aphasia, in which he included both word-deafness and word-blindness, McCready believed that the high degree of intelligence reported in some cases was not borne out by detailed tests, but nevertheless insisted that the defects were not incompatible with normal or even superior intelligence. In a series of fifteen cases of congenital word-blindness, Orton[54] reported superior intelligence in one, average intelligence in nine, mental dulness in one and marginal defectiveness in four. McCready emphasized the fact that the recognition of the congenital word-deafness would save a diagnosis of feeble-mindedness in many cases, and in those already feeble-minded would indicate the method of training to be employed.

Our own patient was perfectly normal mentally; and the investigation of his condition and the study of the cases described in the literature have led us to the following conclusions concerning the mental state of children who suffer from congenital word-deafness.

(1) The mental condition of such children is potentially normal. (2) The fact that they cannot speak or speak only imperfectly, and in addition fail to understand spoken language, often gives the impression to the uninitiated that they are mentally defective. (3) With suitable environment and even in the absence of special education adapted to their particular disability, they have normal intelligence. (4) With a poor environment and education which pays no attention to their disability, they are backward and fail to acquire the knowledge necessary for them to take a normal position in life. (5) Under such conditions they may become imbeciles from deprivation as did deaf-mutes before their condition was fully understood, but such 'imbecility' is entirely due to the fact that their defect has not been recognised and is not the result of the defect itself. (6) In some cases, poor environment and lack of sympathetic understanding may lead to antisocial tendencies and delinquencies. (7) The mental condition in cases of congenital word-deafness depends entirely upon the recognition of the defect, the accident of environment, the presence or absence of training suited to the individual's needs and the placing of him in an occupation which makes use of his abilities and puts as little stress as possible upon his defective functions. It is entirely a matter of diagnosis and suitable education.

THE PSYCHOLOGICAL REACTION AND THE EFFECT ON BEHAVIOUR.

In some cases of congenital word-deafness the psychological reaction of the patient to his defect and to its consequences is of paramount importance.

The psychology of this defect and of associated defects has so far been investigated by very few workers, but in the few contributions in which references have been made to the condition certain important features have been emphasised.

The psychological reactions of 'congenital aphasics' have been discussed chiefly by Orton[55], who recognised certain definite types. Some of the patients are contented, show an apathetic disregard of their handicap and its results, and are apparently quite happy in the condition in which they find themselves. It is possible, however, that this contentment and apathy are due as much to ignorance of the nature of the defect and of its influence upon their future as to an actual adaptation to the situation, which Orton believed to be the case. Our own patient was perfectly happy and contented, and it was only when he was told that when he grew up he would have to work like his father that he revealed the only reaction he had to his defect by saying that he would not be able to do so as he could not speak. Other patients develop a mild paranoid reaction to their teachers, to those whose duty it is to guide or discipline them, and also to society in general. According to Orton, from this reaction there arised behaviour defects, playing truant, petty stealing, the damaging of school property and other delinquencies. Others again feel their situation keenly, realise that they cannot do as well as their fellows, develop a sense of inferiority and show disorders in behaviour. Others again develop what Orton calls "emotional blocking"—a blocking of output when anything is demanded of them. In short, congenital aphasics are apt, especially if emotionally unstable, to fail in their social adjustments if their condition is incorrectly diagnosed, or if they are imperfectly understood and forced into occupations which are unsuitable for them because of their defect.

Brônner has investigated the psychology of special abilities and disabilities in detail. One of her patients, who suffered from a limited memory defect for numbers, had been in court several times for sex delinquencies, was obsessed by sex ideas and had written obscene letters. "It cannot be definitely stated that his lack of ability for certain mental tasks had any direct relation to his misconduct, but, on the other hand, it is a fair assumption that had his school progress been altogether normal, he might have developed good mental interests which could have been sufficiently strong to prevent the growth of the delinquent tendencies which he showed." Another patient, who suffered from a defect for number work and poor powers of forming associations with symbols, had, in spite of good educational advantages, shown a strong tendency to delinquency. The important factor in the development of delinquency in his case was believed to be the choice of an occupation which placed undue stress upon the powers in which he was most defective. A third patient, a case of congenital word-deafness, first came under notice as a problem in behaviour. He was mischievous and was guilty of petty stealing, annoying girls and running away. Brônner was of the opinion that in his case the educational failure due to his

defect had a distinct relationship to his delinqueney. Another patient, suffering from congenital word-deafness, frequently ran away from school and had long periods of idleness during which he stole and otherwise got into mischief.

The tendency to delinquency which undoubtedly appears in some congenital aphasics would appear to be due to a number of factors. It is not the defect itself, but rather its consequence in the form of the patient's relation to his environment, which is directly responsible for the misbehaviour. A good home environment and sympathetic training, even in the absence of recognition of the defect, would appear to save many subjects of the defect from the consequences of antisocial reactions. Conversely, the absence of these advantages would be likely to initiate such reactions at an early age. The effect of attempts at education with other children probably depends upon the patient's reaction to the defect and its consequences. Antagonism towards teachers who fail to understand him, dissatisfaction with his own progress, a sense of inferiority when he compares himself with his fellows, would appear to be the exciting factors in the development of truancy and other delinquencies. Lack of interest in school work and a feeling of discouragement and inefficiency probably lie at the root of the delinquency which occurs in a small proportion of congenital aphasics. "The step from this to truancy is easily made, and from that to more serious delinquency. Educational dissatisfaction is a very frequent beginning of what may develop into a long career of misdeeds." The reaction to a vocation may be equally important. The choice of an occupation which demands the use of faculties in which the individual is deficient and fails to encourage those in which he is proficient leads to discouragement, the inability to hold a situation, the frequent changing from one place to another, and a final drifting into delinquency. A poor and unsympathetic home environment, attempts at education ill-adapted to the individual's needs and the choice of an unsuitable vocation would then appear to be factors of importance midway between congenital deficiency of the type described and antisocial behaviour. Their importance lies in the fact that they can all be corrected in some measure at least. Early appreciation of the presence of a real defect, early recognition of the true nature of the defect, education adapted to the defect and the special abilities of the patient, and finally the choice of a suitable occupation would tend to eliminate the tendency to delinquency which is present in that proportion of congenital aphasics who are emotionally unstable. Although in more stable individuals such tendencies are much less apparent, their reaction to their defect and to society would certainly be improved by the early recognition of their defect and suitable education.

TREATMENT AND EDUCATION.

The treatment of congenital word-deafness consists entirely of appropriate education which in turn depends upon the early recognition not only of the defect which is the obstruction to the individual's career, but also of the abilities

that may be used with advantage in his education. The general aim of all such education must be the socialization of the individual and, to achieve that end, it must be remembered that different individuals, even those suffering from the same defect, need different methods of treatment. Measuring the intelligence of the individual according to a scale such as the Binet-Simon is a beginning to the study of the individual but fails completely to give a correct picture, for as a rule the success of such a method depends upon the correct understanding of oral instructions and it is in the appreciation of these that the subjects of the defect invariably fail. It is necessary to mention this fact because many individuals who present problems in education are studied in the first instance by such methods and are seldom, at such an early stage, investigated completely by ordinary clinical methods. If such methods are used they must always be supplemented by a thorough clinical examination of the patient, a careful analysis of the speech defect and a complete examination of the defects in the other zones of language before recommendations can be made for practical guidance in education. It is well, therefore, to consider what are the chief difficulties the average sufferer from congenital word-deafness has to face in education, and what are the most suitable methods of approach. Education by the auditory route as applied in conventional methods of education is not possible; by the visual route, when applied in the conventional manner by the use of reading, education is very difficult as there is usually present some lack of appreciation of the significance of written and printed symbols. However, when applied by lip-reading and by the use of the patient's power to appreciate form and shape not only by touch but with the eye education will as a rule produce good results. Nevertheless, there remains to contend with some impairment of 'internal speech,' as we have shown in Head's 'Hand, eye and ear test.' Thus it is obvious, however much the defect may appear to be limited to the auditory field and however easy education through the visual and other channels may appear to be, that there are usually complicating defects in these other channels of approach which render education through them much more difficult than would appear at first sight.

The education of the subject of congenital word-deafness has received little attention. By Wilbur it was attempted on the assumption that the defect in language was due to mental deficiency and that the imperfect volition responsible for the defect could be remedied in some cases by patient training; by Hadden and Guthrie in the belief that the defect was almost entirely one of speech; but by no one has it been applied with a full appreciation of all the difficulties involved. The educational problem in all such defects has been emphasized by Brônner in the following manner. The problem is not that of segregation, as in the case of the feeble-minded and the mentally deficient; it is rather adjustment to the social organism. The first step in developing each individual in relation to his potentialities is to recognise that he needs individual adjustment. The second step is to realise the need for educational

diagnosis. Early investigation is essential. "The first and most obvious advantage of early investigation is the saving of time that would otherwise be wasted. The second is the prevention of discouragement and loss of interest. With the consciousness of failure, there is, all too frequently, emotional disturbance leading to the development of a bad attitude either towards the difficult subject or towards school in general. Frequently, the child is subjected to teasing by his comrades or to scolding by his teachers; sometimes friction arises at home as well as at school, and as a consequence of these irritations, antisocial grudges are formed. Because of these conditions, leading to lack of self-confidence and sometimes to excessive mental disturbances, we find that it is often extremely difficult to induce the elder child or adolescent to make any effort under systematic guidance to overcome the defects. Again and again we have seen that, in spite of desire to master the difficulty, emotions may be stronger than ambition."

The education of the patient must be so arranged as to produce the following results.

(1) To secure for him normal relationships with those with whom he comes into contact in his home, at school and in society.

(2) To provide him with the means of communicating with and understanding his fellows.

(3) To enable him to qualify by training for some occupation in which he can engage in a manner commensurate with his abilities.

The methods employed have varied considerably in their details, but as a rule have followed the same general principles.

The child may be treated as a deaf-mute and taught as such. Thomas thought that this was the only rational method of educating the subjects of congenital word-deafness. The advantages of this method are that the patient acquires some means of understanding his fellows and can communicate with them more or less successfully. He is, however, made to associate almost entirely with those who are deaf and thus fails to learn much that he would otherwise acquire by association with those who hear and can speak if he were placed among them as soon as his elementary education had reached a stage which would permit him to do so with advantage.

Hadden adopted a method of education almost identical with that used for deaf-mutes, but employed certain important modifications which added much to the success of his treatment. The patient was made to watch and then imitate the actions of the lips, tongue and teeth required for the production of the various elementary sounds, but it was often necessary actually to adjust the parts by the fingers or forceps. After a time he succeeded in saying most of the alphabet correctly; but he had great difficulty in joining consonants and vowels to make even simple words. Although he had made distinct progress during the seven weeks he was under instruction and could produce with an effort nearly all the sounds necessary for articulate language, his spontaneous

utterances were little altered. It was found that in spite of his improvement under tuition, he persisted in talking gibberish to other patients. It was then apparent that if education in methods of speech was to be successful, he would have to be completely isolated for a time. He was, therefore, isolated and given amusements but forbidden to read to himself or to converse in his own language. Instruction in articulation was continued daily. He was gradually taught to join syllables so as to form words, and then to combine words into sentences. He was made to speak very slowly, as any attempt at speed resulted in a return of his faulty speech. After three months he spoke quite well enough to be intelligible, and had learned to read an elementary reading book.

The same method was adopted with a second patient. In three months, one month of which was in isolation, "he had made considerable improvement, and his articulation became quite intelligible." Afterwards, he made slow but distinct progress though receiving no special guidance beyond that at home and at an elementary school.

Colman strongly approved of the method employed by Hadden, but specified the need for a special instructor to be always with the child until he learned to speak properly.

Guthrie developed the above method. While pointing out that the patient must be taught to imitate by eye what he cannot do by ear, he emphasized the need to use other senses as well. The sense of touch could be used for gutturals by placing the patient's hand on the speaker's throat. By a study of the patient, it could be ascertained which sounds were most faultily pronounced. Monosyllabic sentences in which these sounds occurred both as initials and terminals were used and the patient made to reproduce them by the aid of the eye and touch. It was insisted that the sentences employed should be meaningless, otherwise the patient would learn them with ease in his own speech and cease to take pains to reproduce them carefully. Guthrie also advised exercises to train the visual memory and the cultivation of the ear by systematic musical training.

It is difficult to suggest a general method of education for use in all cases of congenital word-deafness as individual defects and tendencies must be considered as well as the fundamental defect, but the following outline of the principles to be followed might serve as a basis upon which to build up a scheme of treatment for the individual case.

(1) Training in articulation is carried out by sight and touch.

(2) Lip-reading is taught as for deaf-mutes.

(3) The ear is cultivated, as far as possible, by getting the patient to associate the correct sounds used to form words with the speaker's words as appreciated by sight and touch, and also with the sight of the objects named.

(4) Teaching in a class for deaf-mutes is to be avoided if possible and a personal instructor is to be employed, to be in daily contact with the patient

until he has progressed far enough to understand speech and to use spoken language well enough to be intelligible.

(5) If the tendency to use a defective speech or to talk gibberish persists, the patient is to be isolated and kept in silence excepting during the periods of instruction. The tuition during this period of isolation is to be more intensive than in the ordinary case, and isolation is to be terminated only when the patient can and will use his recently acquired methods of articulation sufficiently well in his attempts at conversation to make his speech intelligible.

(6) When the use of speech and the understanding of spoken language have progressed far enough, the teaching of reading and writing by a personal instructor using a similar synthetic method is to be proceeded with. This is necessary because of the possible presence of a complicating defect in the visual field such as occurred in our own patient.

(7) As much advantage as possible should be taken of the patient's power to appreciate shape and form, to remember pictures and to use figures.

Having taught the patient to speak intelligibly, to understand spoken language and to read and write, it is possible to provide him with an education similar to that given to other children. In doing so, the methods to be employed must be so chosen as to make no use of the function in which he is deficient. To do so would simply lead to discouragement and the feeling of failure, together with their undesirable results. Speech, writing and reading improve and become normal according to the accounts of some of the cases in the literature, but there is rarely much improvement in the appreciation of spoken language by ear. Yearsley, however, succeeded to a certain extent in cultivating the auditory appreciation of the spoken word in his patient, and it is possible that in other cases there is some slight dormant ability to understand the significance of sounds, which can be developed to a certain extent by systematic training. As all degrees of the defect probably occur, from a limited inability to appreciate the significance of numbers or musical sounds to a complete inability to appreciate the significance of sounds at all, it is advisable to proceed with measures designed to cultivate the ear as long as possible in order to develop whatever latent function may exist.

The choice of a suitable occupation is next to be considered. This choice resolves itself into the recognition of such special abilities as the patient has in spite of his congenital defect, and the avoidance of any type of work which depends for its successful issue upon the appreciation of spoken language by ear. It is quite as important as, or even more important than, the use of correct methods in the elementary education of the individual. The defect precludes the possibility of success in certain fields of endeavour, and these must be carefully avoided if the patient is to be properly socialized and become a self-supporting and self-respecting economic unit. Vocational education adapted to the special abilities of the individual must follow the orthodox elementary education if the antisocial consequences of his defect are to be avoided.

SUMMARY AND CONCLUSIONS.

1. The condition known as congenital word-deafness is reviewed and discussed.

2. Attention is drawn to the varying nomenclature used since 1867 to describe the condition. The descriptive terms that have been applied to the resulting speech disorder gave no indication of the presence of defect in the auditory field.

3. The literature of the subject is reviewed and the various recorded cases discussed.

4. The clinical features of the defect are: incidence in boys much more frequently than in girls, a familial factor in about one-third of the cases, inability to appreciate the significance of spoken words, slight defects in the appreciation of written and printed symbols, absence of or defects in speech, and defects in writing and spelling resembling those in speech. These features occur in a child whose hearing and other functions are perfectly normal.

5. The condition is due primarily to a defect on a lower level of function than that which results in true aphasia and might be more correctly termed 'congenital auditory imperception.'

6. It is probably due to absence or defective development of the parts of the brain concerned in auditory reception, and the changes are bilateral in distribution.

7. The fundamental defect is inability to appreciate the significance of the spoken word (word-meaning-deafness) and the associated defects in speech, visual appreciation and writing are of the nature of a complicating aphasia of the type described by Head as 'verbal aphasia.'

8. Children suffering from the defect are potentially normal mentally, but because of their difficulty in understanding speech and their inability to express themselves intelligibly may be thought to be mentally defective. In a good environment they can become normal mentally, but in a poor environment and in the absence of sympathetic education they may become imbeciles from deprivation.

9. In some cases the psychological reaction of the patient to his disability, to society and to his vocation, if he has one, may be disordered and lead to antisocial behaviour and delinquencies.

10. Early diagnosis and education are essential. The method of education must be adapted to the individual as well as to the defect. Elementary education must be followed by vocational training for an occupation which makes use of such special abilities as the patient has and places as little stress as possible upon the function which is defective.

11. Because of the widespread distribution of the defects, education through channels other than the auditory is much more difficult than might be suggested by the apparent limitation of the defect to the auditory field.

12. Congenital word-deafness is relatively infrequent and much less

common than congenital word-blindness; but the incidence of the condition in the general population is probably much greater than is suggested by the small number of cases reported in the literature.

13. In children who are slow in learning to speak, backward at school or apparently mentally defective, this condition should be excluded before a diagnosis of mental deficiency or defective intelligence is made.

14. Early investigation and diagnosis are necessary if special education is to produce good results. The later the condition is diagnosed and special education begun, the more difficult and less successful are attempts at the socialization of the patient.

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THE SCHIZOPHRENIC MALADJUSTMENT.*

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Among the maladjustments of man to his environment there are none more dramatic than those found in the field of mental disorders. In this field one meets disorders of human adaptation of the greatest variety. It includes cases where adaptation has been seriously hampered by gross structural brain disease; at the other extreme it includes hysterical cases where, for instance, a paralysis is found with no evidence of any structural damage. Viewed from the standpoint of the adjustment of the individual to the environment, these two extreme types of disorders represent very different problems. In the organic psychoses with gross brain disease, where there may be definite motor defects, loss of memory, inability to deal with situations with good practical judgment, the picture is one of an actual defect, of loss or reduction of function —the loss being intelligible in the light of structural damage, but not having any particular significance or playing any active rôle in the adjustment of the individual to the life situation. It is somewhat different in cases of hysteria where one meets symptoms superficially somewhat similar, such as paralysis, blindness, memory defect, but where the symptoms are not to be looked upon in a negative way as a defect, which impairs the working of the machine, but where they are to be considered in a positive way, as having meaning, or significance, and playing a definite rôle in the adjustment of the individual to the life situation. This hysterical adjustment may receive a judgment of value from others who condemn it as one of inferior quality; at any rate adjustment has been made, which, while socially inferior, may have its own value for the individual. In the organic psychoses the defect has no value for the individual, or if it does have a certain value as in relation to pensions and compensations, that value is a secondary matter and is absolutely irrelevant to the development of the defect. The paraplegia which enables the hysteric to avoid the danger of battle or the effort of industry by means of his apparent invalidism, is part of an adjustment, while the paraplegia of a cord tumour is a mere defect without meaning.

In our study of mental disorders in general it makes a very great deal of difference whether we consider exclusively the principles which dominate the study of the organic psychoses, or whether we utilize the point of view which has thrown so much light on hysteria and the other psychoneuroses. It makes a great difference whether we consider symptoms as defects or anomalies

* Read at a symposium on Maladjustments at the Ninth International Congress of Psychology, Yale University, New Haven, September 1929.

dependent on some structural or toxic damage to the nervous system, or whether we scrutinize each symptom and syndrome as possibly having significance, and as being part of an attempted adjustment to a life situation. The one attitude does not exclude but should supplement the other. A symptom may have its origin in certain structural or toxic factors, while at the same time it may be utilized for adaptive purposes; the severity and duration of the symptom may only be intelligible in the light of the actual situation and of the strivings of the individual.

When the study of mental disorders became possible through emancipation from mediæval ideas of possession the first tendency was to assimilate the study of the symptoms of mental disorders to the study of physical diseases, and to look for their explanation in terms of structural and clinical pathology. Histopathological and biochemical studies have been very illuminating: the symptoms in the organic and toxic psychoses become more intelligible in the light of definite cortical and subcortical lesions, of inflammatory processes and metabolic disorders. Notwithstanding the prestige due to these discoveries and the desire of some workers in these fields to extend their formulations over the general field of mental disorders, it became evident that investigation along these lines had thrown little light on the so-called functional psychoses. Hysteria and the other psychoneuroses had been illuminated when the symptoms were studied in the setting of the adjustment of the individual to the environment, when adequate recognition was given to the complexity of human nature and to the actual significance of the life situation to which the patient had to adjust. The progress made in the study of the psychoneuroses stimulated a reorientation in the study of the functional psychoses, and the stimulus given to this new orientation by the formulations of Adolf Meyer and of Jung on the schizophrenic group has done much to direct the work of the last two decades. While the main principles were thus clearly outlined twenty years ago, the necessity for the painstaking accumulation of well-analysed material still exists. In the field of psychiatry the experimental method has strict limits, and in view of our inability to experiment with the main issues of life it behoves us to gather carefully all we can from the ruthless experiments of nature.

The technical difficulties in the accurate analysis of this material are considerable. The schizophrenic group of psychoses is by no means easy to delimit, and there is no general consensus of opinion as to the exact criteria which entitle a case to be included in this group. In this communication the term schizophrenia is provisionally used in a rather broad way to indicate a group of cases of the greatest diversity as to both symptomatology and course; in general the patient in this group has a formulation of experience, which contains elements so odd and unusual, that they seem completely alien to the structure of the normal mind. This alien appearance may, however, be due not so much to a real lack of kinship with the normal mind, as to the fact that

we are much less familiar with the organization of the latter than we have been wont to assume. The study of problems of the normal adult mind in laboratories of experimental psychology had not revealed the persistence of childhood and primitive modes of experience beneath the conscious processes of the modern adult, nor the full importance of the sex factor with its tributaries from infantile and juvenile sources.

With a fuller appreciation of these latter factors the behaviour and utterances of the schizophrenic seem less meaningless; we realize, however, that in studying the latter we have to understand a new idiom. The idiom is much simpler in some cases than in others; the degree to which it renders intelligible the schizophrenic experience is variable. Even with some knowledge of the schizophrenic idiom the total manifestations may appear to be only in part adaptive and with meaning, and some symptoms seem to have no more significance than defect or irritative symptoms of organic or toxic origin.

A young woman claims that she has two selves, the new self on the left, the old self on the right of her body; she is to be the mother of God; she became pregnant (there was no pregnancy) without coitus, she has had sex relations with God; she has seen and spoken with God.

These beliefs are quite alien to her organized body of knowledge, but represent obviously not merely a defective mode of thought of no adaptive significance or value; the girl has been brought up in a strictly religious family, her acquaintanceship with boys was severely restricted, she has been found unsuited for the religious life. She develops an experience which satisfies her sexual and religious cravings, while she recognizes the conflict between the different forces in her nature.

Is such a surrender to the imaginative satisfaction of cravings and such acceptance of the naive formulation of the two selves possible without a defect of organic or toxic origin? In answering the question one must consider not only the data of the various laboratories but also the facts of the religious life in general. The latter may require critical scrutiny, but they too represent valuable experiments of nature in which personalities of various types have been faced with a great variety of situations.

In the present case the girl, of imaginative nature, steeped in a religious atmosphere, had reacted to the restrictions and values of the social environment with a passive acceptance of real deprivation but with intense absorption in imaginative realization of her cravings.

In other cases the reaction of the patient is not so simple as in this example of indulgence in undisguised imaginative satisfaction.

Thus a woman in the forties, married to an impotent husband, begins to complain that the house is wired with electricity by her husband to annoy her, that the 'movies' demonstrate her life and at the same time use plots plagiarized from her, that spirits of dead artists commune with her. She berates a woman because the latter makes a gesture signifying "you take her, she's yours!"

In this case the experience seems more remote from the structure of our everyday experience than that of the religious girl referred to. It is true that the feeling of the presence of the spirits of the great artists of the past is sufficiently close to the religious experience; but the distorted view of the environment ("wired with electricity"), the complicated interpretation of the 'movies,' and of the trivial gestures of strangers, are not simply related to any familiar emotional craving. One might be inclined, therefore, to say that the distorted structure of this patient's experience was only to a slight degree intelligible as an adjustment to the actual life situation, and on the whole was a meaningless disturbance of the thought functions.

While the latter is possible, it is well to do justice to the actual nature of the symptoms. The patient's reference to the house being all maliciously "wired with electricity" by her husband, may be her way of meeting the actual feelings due to the unsatisfactory marital relations; instead of thinking clearly and realistically about the disturbing sexual factor, she finds it more tolerable to think of it in less direct and less adequate terms, in terms of electricity, for which she has no personal responsibility. Such a solution of a problem in sexual adjustment is sufficiently familiar. Such an interpretation can be sufficiently documented from a wide clinical material so that in the individual case it loses its arbitrary and a priori appearance.

The interpretation of the 'movies' and of the gestures of casual strangers demands a detailed analysis of the structure of experience and the recognition not only of repressed factors of emotional importance, dissociated from the rest of the conscious life, but also of the persistence in the adult of primitive and childlike modes of thought (Jung, Storch). It is one of the present tasks of psychiatry to collect material which will show to what extent these primitive and childlike modes of thought lie dormant beneath the surface of ordinary adult thought, as the primitive mass-reflex lies unnoticed in the highly integrated activities of the intact spinal cord.

The two cases cited above present a comparatively simple clinical picture; in other cases one finds a much deeper disturbance of the organization of thought and behaviour with complete neglect of logical canons, great fluidity of concepts, perplexing statements without propositional form, neologisms, with equally marked disorders of behaviour. Under stress and strain human nature can go very far in the way of deviation from rational thought and action in an evasive, imaginative and regressive direction, with the reactivation of discarded modes of experience and with the disguised manifestations of crude and repressed instinctive tendencies, where there is no unequivocal evidence of any primary structural damage or intoxication. In many cases the disturbance of conduct and utterances is so profound that some take for granted that there must be some definite break and some irreparable damage to allow of such a degradation of human behaviour, and yet one may see the return from such a condition to complete previous normality with no indication of anything having been permanently lost.

While the schizophrenic disorder is to a large extent intelligible as the reaction to the life-situation, the latter need not be of any dramatic quality. In the history of the patient as recorded one may note no outstanding event or situation which seems to involve undue strain, and conditioning factors of importance in early life may have escaped the notice of those near to the patient.

When one scrutinizes the symptoms of the psychosis and when one reviews with the patient the various steps in his experience one appreciates better the rôle which the actual life situation has played in the whole development. While banal as seen from the outside it may have been the very stuff out of which the special personality of the patient was likely to weave the abnormal fabric of its experience.

The adult environment may be of very normal appearance and the early moulding forces of the patient's life may seem to have been little different from those of his fellows, but the unobtrusive forces of the environment may have subtly and profoundly influenced his adjustment to the insistent demands of sex, to the bond between parent and child, to the opinion of his fellows, to the spiritual versus the material values of experience, and may have modified the vulnerability of the personality in relation to the tests of the adult life. This vulnerability, partly determined by congenital endowment, is also influenced by a long series of situations and experiences, the significance of which is apt to be overlooked.

The overt series of reactions, which constitute the psychosis, is often but the later stage of a maladjustment which stretches far back to the childhood of the patient. The study of the etiology of a schizophrenic psychosis is the study of the action and interaction of an individual and his environment over a long period, and throws light on many phases and varieties of human maladjustment.

Short Notes and Clinical Cases.

A CONTRIBUTION TO THE PATHOLOGY OF THE LATER MANIFESTATIONS OF ENCEPHALITIS LETHARGICA: INVESTIGATION OF A CASE.*

By

F. E. REYNOLDS AND JAMES K. SLATER, EDINBURGH.

INTRODUCTION.

THE object of the present investigation was two-fold: firstly, it was desired to ascertain whether an acute attack of encephalitis lethargica from which recovery took place gave rise to organic destruction of nervous elements with consequent changes in the neighbouring neuroglia; secondly, it was hoped to gain some indication whether the well-recognised progressive later manifestations of disturbance of the nervous system are to be explained by a slowly progressive lesion initiated by the acute attack.

The present case was particularly suitable for the investigation since the patient had lived some eight years after a typical acute attack.

CLINICAL REPORT.

J. J., spinster, aet. 59, was admitted to the Royal Infirmary, Edinburgh, on February 12, 1921. We are indebted to Professor Edwin Bramwell for the following clinical notes.

History.—The details of the patient's illness were ascertained from her sister. Some three weeks previous to admission there had been a gradual onset of drowsiness and lassitude; the drowsiness became so pronounced that, five days later, the patient had to take to bed, and she slept almost continuously; when sufficiently stimulated she could be roused, although from this time it was necessary to feed her.

The patient continued more or less in the same state up to the time of admission to hospital; throughout, when roused, she was always able to appreciate what was said to her. About a week before admission she began to complain of occasionally seeing double and of indistinctness of vision.

On admission. The patient was extremely drowsy, but could be wakened; she was able to give intelligent answers to questions, but spoke in a slow, monotonous voice and only after a pause. It was observed that the face was immobile and devoid of expression. The right pupil was larger than the left, but both pupils reacted to light and on accommodation; the patient was unable to converge especially with the right eye, and could not look upwards beyond the horizontal plane. The abdominal reflexes were not elicited; the plantar response was flexor on both sides. No abnormal signs other than those mentioned above were detected in any system.

* From the Laboratory of the Scottish Asylums' Board and the Department of Clinical Medicine, University of Edinburgh.

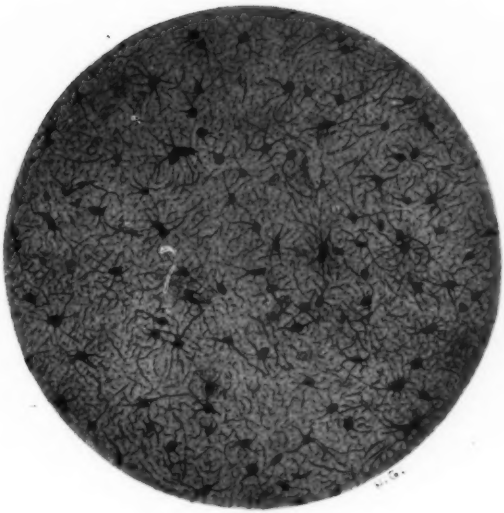

Fig. 1. Preparation from the mid-brain showing great proliferation of astrocytes, some of which are large and in various stages of amitosis. (Frozen section treated by Cajal's gold chloride sublimate method).

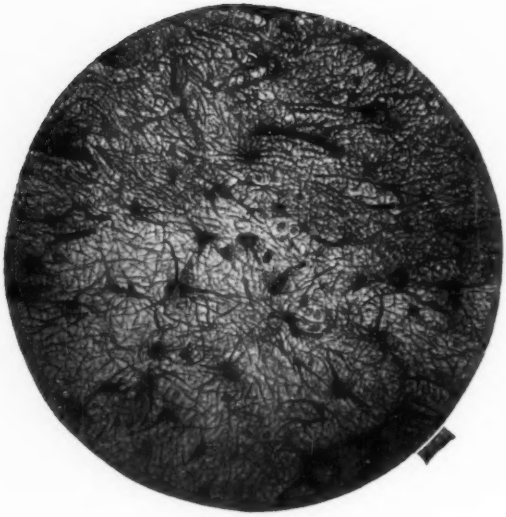

Fig. 2. Preparation from the mid-brain. In the upper part of the section, dense gliosis is present. In the lower part, the fibrous elements are less numerous and some of the astrocytes are still in a phase of vegetative activity. (Frozen section stained by the Kristallviolett method).

Six days later (February 18, 1921) a lumbar puncture was performed. The cerebro-spinal fluid was clear, not under tension, and contained 40 cells per c.mm., the majority of them being reported as small lymphocytes.

Progress.—The patient remained in hospital for five weeks. During the first three weeks the temperature was occasionally rather above normal, but thereafter pursued a perfectly normal course. The drowsiness, the immobility of the face, and the slowness of speech passed off gradually. Before she was discharged, all movements of the eyes could be well carried out, but the right pupil remained slightly larger than the left.

Shortly after leaving hospital, she developed involuntary movements of the legs; they were intermittent, of varied intensity, of an irregular compound nature, absent during sleep, on walking, and on making other voluntary movements.

In January, 1922, one year after the acute attack, the patient was admitted to the Royal Edinburgh Hospital for Incurables; but after five months she was discharged from the Institution, since her condition had become no worse and she no longer required indoor treatment.

On leaving the Royal Hospital for Incurables, the patient went to live at Queensberry House, Edinburgh—a home for old people. She there came under the care of Professor William Russell.

For the first four years of her stay in the Home, she was able to go about the buildings and in the gardens. During this time it was observed that, when walking, she tended to trot, and held the arms in a flexed position across the trunk. Further, her face became expressionless and her condition one of apathy.

These signs of Parkinsonism gradually progressed and necessitated the patient spending more and more time in bed; after 1926 her condition was such that she was disinclined to get up. Eventually her death was due to inanition.

During the acute attack, the symptoms indicated that a definite focus of damage had occurred in and around the nuclei of the third cranial nerve, and the midbrain was selected, therefore, for investigation.

After autopsy, the portion removed was fixed for ten days in formol-ammonium bromide solution, and then frozen sections were made in a plane at right angles to the long axis of the midbrain. Sections were impregnated by Cajal's gold chloride sublimate method and by Gross-Bielschowsky's method: a third series was stained by the Kristallviolett method for neuroglial fibres.

REPORT ON THE MATERIAL EXAMINED MICROSCOPICALLY.

Throughout the midbrain, areas were present in which great proliferation of astrocytes had occurred: for the most part the cells were fibrous in type.

Many of the proliferated astrocytes had regressed and formed areas of dense gliosis. On the other hand, not only were there large astrocytes with thick processes and nuclei rich in chromatin, but the cytoplasm of some contained two nuclei; further, throughout the areas of gliosis, but especially in certain parts, some of the large vegetative astrocytes occurred in pairs and small groups. Thus the stages of amitotic division were present. (Figs. 1 and 2.)

Among the areas in which gliosis was especially pronounced were the regions of the nuclei of the third nerves, and the nuclei themselves had been encroached upon by the astrocytic proliferation.

COMMENTARY.

As a means of elucidating the nature of any disease, morbid histology has certain very definite limitations. It is of positive value in this connection only if changes in the structure of the cell are demonstrated or the anatomical relationship of the cell to its environments is shown to have been altered. Further, it can fulfil this purpose only if the vital processes which have brought about the changes can de deduced from the altered form and arrangement.

Microscopical examination of the tissues may reveal the results of acute disease. But unless the sequelæ are causing alterations in the function of the body cells at a later time, the condition is one of repair and not of disease. On the other hand, before a more or less longstanding condition can be designated a chronic disease, it must be producing an effect, either local or general, on the individual cells and tissues. This effect is naturally progressive.

We have described histological changes in the present case, and we shall argue later that they are not only consequent on an acute condition, but that they represent an instance of a chronic progressive lesion. Before doing so, however, it is necessary to consider the significance of the proliferation of interstitial cells which occurs so frequently in morbid processes. Our views on this subject have been stated briefly in a paper recently published by us (SLATER AND REYNOLDS, "A study of a case of cervical glioma," *Brain*, 1929, LII, Part 4).

Throughout the body, whenever destruction of tissue occurs, the interstitial supporting cells proliferate. In the paper referred to, we suggested that the products of lysis of the dead tissue stimulate the reproduction of the cells. The phenomenon, however, may not be the result of actual destruction but may be consequent upon damage to the tissue of such a degree as to give rise to abnormal metabolic products of a potency sufficient to provide the stimulus for proliferation of the neighbouring cells. Again, direct damage to the interstitial cell itself may cause it to divide, provided the damage is not so severe as to destroy it. This is a natural corollary to the fact so commonly observed in tissue which is undergoing necrosis, viz., that one of the last vital acts of a dying cell is to attempt to reproduce. Even in necrosing nervous parenchymal cells—cells which in the process of specialisation have lost to all intents and purposes the power of reproduction—we have seen occasionally two nuclei; this observation can be explained only on the fact that the dying cell has attempted to reproduce itself, and is in keeping with the fundamental principle of phylogenetic survival. The reproduction of cells now being considered occurs not only in those states which are commonly designated 'disease,' but in conditions of trauma; the law enunciated is merely the statement of a fact fundamental to healing and repair.

With this conception of the basis of cell proliferation in relation to damage and destruction of tissue, we can attempt to translate the histological data in the present case into the processes of which they are the expression.

It may be taken as proved that acute encephalitis lethargica is a condition due to a 'virus.' In the present instance the virus caused damage and destruction to the tissues of the midbrain, whether parenchymal or interstitial, with consequent proliferation of the interstitial, viz. the neuroglial, cells capable of reproduction—the astrocytes.

The histological picture, however, does not show astrocytes which have proliferated merely during or immediately subsequent to the acute attack and then, having developed fibres, have passed from an active to a passive or quiescent stage. Some eight years after the acute attack, large astrocytes with thick processes are present; some of them are arranged in pairs and small groups, and some even have two nuclei in the cytoplasm. These cells are typically astrocytes in an active phase of reproduction. This means that damage or destruction is still going on and it is justifiable to presume that the process has been continuous. Moreover, this supposition is supported by the widespread distribution of the gliosis present at the time of death; judging by the scar formed after repair of traumatic lesions of the central nervous system and having regard to the types of proliferated astrocytes in the case now published, the appearance cannot be explained by the occurrence of an acutely produced lesion eight years ago followed by a period of complete quiescence with a subsequent and very recently initiated active process.

Again, a theory which advanced a recent progress beginning de novo could not explain adequately the nature and source of the factor responsible. It must be concluded, therefore, that in addition to the acute process of eight years ago, the present case presents a chronic and continuously progressive condition.

It remains now to consider how such an interpretation of histological facts coincides with the clinical progress of the patient.

At the time of the acute attack attention was directed to the midbrain by the occurrence of certain prominent signs. Apart from the problematical significance of drowsiness, lassitude, and want of expression, a lesion of this part of the central nervous system was shown by inability to converge or to look upwards; this was supported by the pupils being of unequal size. As we have argued, the lesion in the midbrain at that time consisted of damage to certain parenchymal structures, and most probably of destruction of some of their elements. Further, it has been suggested that damage or destruction of interstitial cells occurred. Since the patient recovered the power of ocular movements, however, the destruction and damage to the nerve cells concerned were not of such a degree as to prevent an adequate number recovering sufficiently to fulfil normal ocular functions. Consequent upon the acute lesion, proliferation of astrocytes occurred; and, fibres having been developed in their cytoplasm, they regressed, areas of dense gliosis remaining. Shortly after the acute attack, the patient developed involuntary movements of the legs; these were due undoubtedly to central irritation, but there is nothing

in the case to localise the site. Nevertheless, these symptoms were of value as indicating that a disturbance of function was continuing. Some time later, the signs and symptoms typical of Parkinsonism gradually became manifest; they progressed until ultimately the patient died of inanition.

Microscopically, not only was there found gliosis due to the lesion produced by the acute attack eight years ago, but also continuous glial proliferation had occurred up to the time of death. The continuous proliferation was due to the persistence of the virus in the tissues and was not a direct result of the damage induced by the acute attack. During the chronic stage, the effect of the toxin was much less intense than during the acute onset, but whether this was due to a numerical decrease or to a lessened pathogenicity of the virus or to both factors is unknown. A partial equilibrium between the virus and the tissues was established, but this was never complete; throughout the entire course of the disease, the infective factor predominated.

It will be seen, therefore, that the clinical course of the disease exemplified by this patient may be correlated exactly with the pathological process deduced from the histology. Both the clinical and histological phenomena are those of a slowly progressive disease following on an acute disturbance of function, with damage and destruction of parenchymal tissue.

SUMMARY.

The case was one of acute encephalitis lethargica followed by progressive Parkinsonism. The histological examination showed not only that gliosis had occurred as a result of the acute attack, but that astrocytic changes were active up to the time of death. The pathological process and the clinical course of the disease are shown to be parallel.

In conclusion, we desire to express our thanks to Professor William Russell for the opportunity given us of examining the case and for the notes on progress during the time the patient was under his care. We are indebted to the Medical Research Council for a grant towards the cost of the research and to the Carnegie Trust for financial assistance in publishing the illustrations.

Editorial.

THE PSYCHIATRY OF CHILDHOOD.

IT is exceptional to meet a psychoneurosis in an adult who has not exhibited neurotic traits in childhood or early adolescence ; so exceptional that when an adult patient of this type denies such traits it is very difficult to believe him. Similarly the numerous studies of persons who have developed a psychosis show a remarkable incidence of morbid personal characteristics at an early age. The evidence in this direction is so impressive for certain psychoses, especially schizophrenic and paranoid psychoses, that one theory of their etiology regards them as the end-results of a progressive habit-deterioration. The practical conclusion is clear enough, but communities and even psychiatrists have been peculiarly slow in applying it. It is curious that some modern developments in psychopathology have been partly responsible for the delay. The paradox depends upon what may be called the endogenous bias of the most influential, and especially the Freudian, schools of morbid psychology. They have emphasised the essentially endopsychic nature of psychological conflict, at the expense of the pathogenic significance (as others think) of the struggle between the individual and his environment. Environment is allowed to have a part only when introjection in abstract and idealised form has been hypothesised and the battleground changed from the surface of contact of personality and environment to a locus deeply within the psyche. Thus, in the Freudian conceptions, all the important pathogenic factors are those that produce conflict between the Ego, the ego-ideal, and the instinctive tendencies subsumed under the title of the 'Id.' It is hardly admitted that frustration of the self-preservative instinct by environmental factors can lead directly to a mental illness. This peculiar self-imposed restriction is however compensated to some extent by the Adlerian and Jungian schools.

No one who has worked with children, nervous in any sense, can fail to be struck by the immense influence of environmental factors, including not only the immediate family, but

also companions, school, teachers and the like, upon the production of symptoms; and similarly by the comparatively speedy and sometimes dramatic results it is possible to produce in children by treatment, including manipulation of the social surroundings. The part played by such influences as imitation and suggestion appears to be very great, and hints strongly that factors of this kind, which were so much stressed by the older psychologies, and which have been considerably neglected of late, may yet come into their own in the domain of morbid psychology. Such old-fashioned formulations as the need for outlets for self-expression, the importance of incentives and ideals, of habits, social contacts, games, and the like, have a direct application in the treatment of children's neuroses. There remains a residue in which more recondite psychopathological theories may be relevant but their application is another matter. Such psychoanalytic studies of neurotic children as we have had, especially those of Klein and Anna Freud, smack of the methods enployed in the notorious case of Little Hans, and much confirmatory work is required before full acceptance of the findings can be granted. In this particular direction it should be possible from the study of children to arrive at more definite conclusions as to the relative importance of various instinctive tendencies of the child, and especially of the relative importance of self-preservative and sexual instincts in the production of symptoms; while the true significance of infantile sexuality may be more certainly gauged. From a wider standpoint, the extent to which traits of personality are inborn, the manner in which they are acquired, the role of example and imitation, witting and unwitting, the application of Pavlov's conceptions to the theory of the genesis of symptoms, and many similar problems call urgently for investigation. A field of first-hand study offers itself here, and should afford advantages over the approach to psychopathological questions through the dim historical stratification, with its mingled fossils of fantasy and recorded fact, exhibited by the adult mind. Nor can the physical aspects be neglected, especially the interrelation of biochemical deviations and such partial phenomena as enuresis, night-terrors, and some tics.

One of the great lessons that has yet to be driven home in this country is the close association between psychiatry and the public health, especially in the field of preventive medicine, of which a large and practically the most important part is that relating to children. Through such widespread social

factors as heredity, housing, poverty, ignorance, education, and employment, runs the thread of causation of nervous illness in children as well as in adults. Social inefficiency is in most of its instances homologous with nervous illness; while in children nervous illness frequently indicates a fault in the social environment. More concisely, in adults nervous illness produces social inadequacy; in children, it is commonly the result of social inadequacy, but here the inadequacy resides in the thing experienced rather than in the experiencing subject. If the inefficient adult is the child neurotic grown-up, then in the ultimate analysis of both instances the early environmental factors must bear a heavy responsibility.

The organisation of psychiatric work with children presents a problem of an administrative kind. The close affinity with social conditions demands an efficient intelligence service, for which a specially trained type of worker, the psychiatric social worker, has already proved indispensable. Liaison must be maintained also with the various societies and associations which handle children, and also with the schools. Obviously teachers cannot as a rule find time to devote to close individual observation of their charges; but the data of scholastic reports are very useful from the angle of intelligence and capacity to profit by education, while experience has shown the considerable value of observations made by an intelligent teacher on the behaviour and temperament of pupils. For the more accurate assessment of intellectual capacities, both of a scholastic and practical kind, and consequently also for guidance in the choice of a career, a person trained in the application of mental tests is of great value. A psychiatrist, a pediatrician, a psychiatric social worker, and a psychological tester are therefore the desirable members of a team for dealing with nervous or difficult children. At present, the tendency is for 'Child guidance clinics' to be established as clinics independently of hospitals. This means from the medical aspect a certain amount of isolation; it means increased difficulty of access to general and special medical departments, including especially organic neurology; it removes from the experience of the embryo medical man the most promising field of psychiatry, just at the time when psychiatry is being laboriously brought nearer to general medicine; and it misses the advantages of the prestige and tradition of hospitals in treatment and research. Whether the advantages of independence outweigh the disadvantages remains to be seen.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[148] **The rudimentary parietal eye of mammals** (Recherches sur l'existence d'un oeil pariétal rudimentaire—le corpuscule pariétal—chez les mammifères).—K. H. Krabbe. *Kgl. Dansk. Vidensk. Selskab.*, 1929, viii, 1.

In the embryo of certain mammals (horse, dog, hare, etc.), Krabbe has found a small group of rounded and oval cells in the centre of the external aspect of the commissura habenulæ. This little 'organ' is situated immediately above the pineal body, behind the posterior wall of the suprapineal recess, and is distinct from the 'prepineal body' of Favaro and others. To this rudimentary structure he proposes to give the non-committal name of 'corpuscule pariétal'; he considers it is a homologue of the parietal eye of *petromyzon*, teleosts, and saurians.

J. V.

[149] **The cerebral circulation.**—Stanley Cobb. *Amer. Jour. Med. Sci.*, 1929, clxxviii, 528.

This paper summarises recent work at the Harvard Medical School on the question of the vasomotor control of the cerebral circulation. The following experiment is described in detail:

The cervical sympathetic in the cat having been severed one hour to six days previously a dye was injected intravenously. Sections from each hemisphere were then taken and the area occupied by injected capillaries in each was measured. It was found that on the side of the cut sympathetic nerve there was a very considerable increase of injected capillaries. In other experiments pial vessels were observed to dilate on vagal stimulation and to contract on sympathetic stimulation; these changes are evidently independent of changes in the systemic arterial pressure. Cobb comes to the conclusion that vascular spasm is, as a result of these experiments, a much more reasonable proposition than it was thirty years ago.

This paper is of value to anyone interested in the subject as it epitomises work done and gives a fair number of references to recent work on the subject.

E. A. C.

[150] **The cerebral circulation ; the action of histamine.**—H. S. Forbes, H. G. Wolff, and S. Cobb. *Amer. Jour. Physiol.*, 1929, lxxxix, 266.

Vessels of the brain react to histamine (injected intravenously) according to the anæsthetic employed ; under ether they are already dilated, and with histamine either do not enlarge further or sometimes become narrower, with fall of spinal fluid pressure. Under amytal they dilate after histamine, and fluid pressure rises. Local application of the drug to the surface of the brain always results in dilatation of pial vessels, without influence on intracranial or systemic vascular pressures.

Chemical changes in the blood are often more powerful than alterations in blood pressure as a means of regulating the calibre of pial blood vessels in mammals.

J. V.

[151] **Cold stimulation of a peripheral region and the excitability of the cerebral cortex.**—Attilio Rizzolo. *Brit. Jour. Med. Psychol.*, 1929, ix, 258.

In general it may be stated that given a temperature sufficiently low and allowing the factor of time to intervene, cold stimulation of a peripheral region of the body first increases the excitability of the part of the cerebral cortex corresponding to the region stimulated and later decreases it. The effect of the stimulation is not only on the corresponding cortical area but also on other parts of the same or opposite side of the cortex ; the effect is always more marked on that corresponding to the region stimulated.

C. S. R.

[152] **The thalamocortical circulation of neural impulse.**—George G. Campion. *Brit. Jour. Med. Psychol.*, 1929, ix, 203.

In reviewing some of the work of Richard Semon and of Head and Holmes, the writer concludes that (1) the 'engrams' or 'engrammic systems' of Semon are identical with the 'neural schemata' of Head, these terms or phrases both denoting systems of neural paths between different areas of the brain—paths which have been developed and become functional in the course of ontogenic life history. (2) These 'engrams' or 'schemata' furnish the neural correlates of the multitudinous psychological elements which, when integrated into a more or less coherent aggregation of mental symbols or concepts, constitute what we ordinarily call 'knowledge' and which in their interactions and ever-changing functional relations constitute what we ordinarily call 'thought processes.' (3) A cursory consideration of the ordinary phenomena of 'memory,' 'reverie' and the different phases of 'ratiocination' makes evident the need of a means of activating the multitudinous system of 'engrams' or 'schemata' from some central source quite apart from any stimuli

from the sense organs or receptors. (4) The central source of power to maintain a continuous stream of neural impulse, ever changing in the pattern of its flow through the multitudinous system of 'engrams' or 'schemata,' is furnished by the thalami. (5) The return paths from cortex to thalami—the corticothalamic paths—are means of maintaining a continuous circulation of neural impulse between thalami and cortex, thus keeping in an active state those 'engrams' or 'schemata' which form the neural correlates of the mental symbols or concepts which may be employed in any particular phase of consciousness through which the subject may at any time be passing.

At present this must only be regarded as a working hypothesis but one which subsequent research may convert into an established theory.

C. S. R.

[153] **The increase in muscle tone following stimulation of the sympathetic nervous system.**—Norman D. Royle. *Med. Jour. Australia*, 1929, i, 740.

According to the author's claim, stimulation of the abdominal sympathetic trunk, by the direct application of solutions of adrenalin (1 in 10,000) is followed by a very transient increase in muscle tonus. Stronger concentrations (1 in 1,000) are succeeded by flaccidity in the limb supplied by the sympathetic trunk. The changes in tonus and posture are much more evident in the fore than in the hind limbs, a fact which is attributed to the influence of stretch. Deep ether narcosis appears to prevent the increase of tonus after sympathetic adrenalinization. Royle refutes the suggestion that altered vascularity is responsible for changes in muscle tone after ramisection, and in one experiment he clamped the femoral artery immediately after the adrenalinization of the sympathetic trunk. Tonus was unaltered by the interference with blood supply.

M. C.

[154] **The influence of the proprioceptive system upon the crossed extensor reflex.**—J. Pisuner and J. F. Fulton. *Amer. Jour. Physiol.*, 1929, lxxxviii, 453.

The experiments (on cats) here recorded bear on the question of the influence of posterior root section and of rotation of the head upon the responses of the quadriceps muscle of decerebrate animals to ipsi- and contralateral break-shock stimuli applied to the sciatic nerve.

Under constant conditions of the neck and labyrinthine proprioceptive fields, section of the posterior root supply to the quadriceps diminishes the latency and increases the rate of development, size, and duration of the crossed

extensor reflex. Evidently posterior root section removes proprioceptive inhibitors which normally are activated by sudden contraction.

Rotation of the head (chin towards the recording muscle) has the same effect on the crossed extensor reflex, with the exception that it diminishes markedly the rate of relaxation of that reflex.

This same head rotation reverses the reflex effect produced by a single break-shock applied to the ipsilateral sciatic nerve. Similar reversals occur in the response of the knee flexors.

From these experiments it seems clear that neck and labyrinthine proprioceptors influence spinal centres. Some impediment which hinders the passage of impulses across the cord is removed by rotation of the head. Presumably this impediment is due to some form of continuous inhibitory stimulation. The same release which facilitates the passage of impulses from one side of the cord to the other also converts the ipsilateral extensor response from one predominantly inhibitory to one predominantly excitatory. This reflex reversal seems to be brought about through some activation of some higher centre of the nervous system than the spinal.

J. V.

NEUROPATHOLOGY.

[155] **The pathological conditions of the brain in infants** (Le encefalopatie infantili)—L. Magni. *Riv. di pat. nerv. e ment.*, 1928, xxxiii, 231.

In this excellent review of a complicated subject the author points out the confusion which has arisen from cross classifications in the past. He proposes that we should divide the groups into (1) antenatal, (2) natal and (3) postnatal.

Leaving out of account familial conditions such as amaurotic family idiocy and hereditary family ataxy, many cases of paraplegia seem to show a family incidence. It is necessary, however, to be certain that such cases are true abiotrophies and not coincidental lesions at or after birth. At the same time there is evidence that hereditary factors play a part in determining other lesions later in the development of the infant.

Specific intoxications (lead, etc.), chronic alcoholism, and syphilis in the parents are important factors especially if they occur in both. The author, unlike some other observers, finds definite signs of syphilitic heredity in about 15 per cent. of cases only.

The post-natal infections are confused and comprise many conditions whose etiological nature is obscure. The author thinks that it is only rarely that it is possible to distinguish between primary or secondary lesions either clinically or by pathological examination.

Among the more clearly defined clinical syndromes the author includes (1) infantile cerebral hemiplegia. He points out that if this comes on after the age of eight it is in all respects similar to the hemiplegia of adults. In younger

children it is often accompanied by Jacksonian fits and contractures may be associated with flaccidity. The tendon reflexes may be less altered than might be expected, but synkinesias are frequent. The limb may be small not so much from atrophy as from want of growth. Varieties of this condition may be met with, ranging from a condition presenting athetosis without contracture or atrophy, without modification of reflexes and without grave intellectual defect, produced by a lesion chiefly subcortical, to a condition with contractures, exaggeration of reflexes, epilepsy, and serious intellectual disturbances but without athetosis, representing a lesion chiefly cortical in extent.

2. Cerebral diplegia (Little's disease). This is a somewhat confused group of all degrees of severity both intellectual and motor, with or without fits or involuntary movements. The lesions may be defective development, sclerosis, or more or less serious hæmorrhage. The author considers that cases of lower limb paralysis with completely normal intelligence and upper limbs should be included in this group. He does not favour the separation of a group of double hemiplegias (in which the upper limbs are more seriously affected) from the general category of cerebral diplegias.

3. Pseudobulbar cases affecting the bulbar nuclei and often accompanied by extrapyramidal motor lesions.

Among extrapyramidal conditions the author distinguishes clinically between those reputedly due to lesions in the pallidum (archistriatum) producing rigidity, and those due to lesions of the neostriatum producing involuntary movements, athetosis and chorea. However he points out that pathologically such a distinction cannot be upheld. The clinical types discussed are :

1. The ' double athetosis ' of Vogt, with generalized athetosis and rigidity of the limbs but without constant changes in the reflexes. This condition which is congenital tends to improve, especially after the first year. The lesion is in the putamen and caudate nucleus.

2. ' Double athetosis ' with spastic diplegia. This may arise after birth and the pyramidal system is obviously involved. The lower limbs are contracted and walking is impossible. The site of the lesion is inconstant but involves the pallidum.

3. Congenital chorea. In these cases, which are rare, choreic movements occur from birth but the nature of the lesion is very uncertain.

4. Wilson's disease. Hepatico-striate degeneration characterized by rhythmic tremors, interference with swallowing and speech, rigidity of muscles and later contraction and mental deterioration. It is a progressive and fatal disease. The lesion consists of a bilateral degeneration of the lenticular nuclei and cirrhosis of the liver. The pseudosclerosis of Westphal-Strümpell is so similar that it may be included with Wilson's disease though some claim that a pigmentation in the former condition distinguishes the two. The condition is familial and probably an abiotrophy.

5. Torsion spasm. This may occur alone or as part of another syndrome and consists of a dystonia of the muscles of posture.

6. An atonic form in which atonia of certain muscles accompanies a cerebral lesion.

Various formes frustes are found which may be due to arrest of development, especially when we consider the hypertonus and inco-ordinated movements of the newborn infant.

R. G. G.

[156] **Contribution to the pathological anatomy of chronic chorea** (Contributo all' anatomia patologica delle coree croniche).—C. BELLAVITIS. *Riv. di pat. nerv e ment.*, 1928, xxxiii, 407.

AFTER reviewing the literature the author describes three cases of chronic chorea—one of Huntington's chorea, one of doubtful origin, and one following an acute attack. While he found the main lesions in thalamus and corpus striatum, others also occurred in the cortex, especially in the fronto-rolandic area. In all the cases the liver was the seat of pathological change of one kind or another. He concludes that Huntington's chorea and chronic chorea following an acute attack seem to be different diseases both anatomically and clinically; the first is a disease of the nervous system with few signs of inflammatory reaction and having affinities to systemic nervous disease; the second shows lesions in all parts of the system and is more easily explained as resulting from an inflammatory process.

Efforts are still made to maintain the hypothesis of a purely striate lesion to account for all forms of chorea, including the hereditary variety; but it seems necessary to suggest a less restricted theory, one which includes cortex, cerebellum and thalamus among the sites of lesions. In Huntington's chorea there is a complex of lesions in the striate and fronto-rolandic areas.

R. G. G.

[157] **Endarteritis of the smaller cortical vessels in severe infections and toxæmias.**—N. W. WINKELMAN and J. L. ECKEL. *Arch. of Neurol. and Psychiat.*, 1929, xxi, 863.

SEVEN cases are described of changes in the brain in severe infections and toxæmias. These conditions include typhoid fever, acute rheumatic fever, toxæmia of pregnancy, erysipelas, Hodgkin's disease, chronic tuberculosis and an undetermined toxic condition. The clinical picture consisted of delirium, at times of a most severe degree, with increase of psychomotor activities such as restlessness, muscle twitching and even convulsions; hyperæsthesia, visual and auditory hallucinations and meningeal irritative signs were at times present.

Pathologically, the predominant change occurred in the small cortical vessels with resultant secondary manifestations in the brain substance. The lining cells of the small vessels showed swelling and proliferation, with formation of new vessels. Scattered throughout the cortex were small microscopic areas of partial or complete softening resulting from cutting off of the blood supply by the swollen endothelial cells. Cloudy swelling in the ganglion cells was a universal observation. Reactive glial formation occurred. The process was not an encephalitis.

In the chronic stages, regression occurs. The endothelial cells of the vessels become shrunken and atrophic: the media becomes swollen and hyalinised and the adventitia fibrous. Organisation takes place in the areas deprived of their blood supply.

Recent work from the chemical standpoint of altered pH of the blood in toxæmias and infections may well account for the pathological manifestations. Therapy directed toward this altered chemical state is indicated.

R. M. S.

[158] **The pathological anatomy of polyneuritis—systematised motor variety** (Sull'anatomia patologica della polineurite, varieta sistematizzata motrice).—O. Rossi. *Archiv. dell' Ist. biochem. Ital.*, 1929, fasc. 1.

The case is described of a woman of 51, whose condition was one of progressive motor weakness of the extremities, especially the lower, with increasing amyotrophy but without any objective and only trifling subjective sensory change. The deep reflexes were abolished. Apparently the symptoms came on after a febrile illness diagnosed as typhoid. After a steadily progressive course of about two years death ensued from myocarditis.

Pathological examination showed an advanced state of parenchymatous peripheral neuritis, coupled with pronounced interstitial change. Similar alterations considerably less in extent and degree were found in the spinal roots, while the cells of the ventral horns were practically normal. Investigation of the mixed peripheral nerves showed a distinct division in the bundles between normal and degenerated fibres. The healthy (presumably sensory) fibres were situated as a rule to one side in the fasciculi, and did not surround the degenerated motor elements.

S. A. K. W.

[159] **Myatonia congenita** (Ueber Myatonia congenita).—Max Bielschowsky. *Jour. f. Psychol. u. Neurol.*, 1929, xxxviii, 199.

This fine histopathological study is based on the examination of no fewer than five typical cases of Oppenheim's myatonia congenita, and merits careful attention. A large number of workers have persisted in arguing for the

anatomical identity of the condition with the infantile spinal muscular atrophy of Werdnig-Hoffman, but Bielschowsky shows that this view is fundamentally incorrect and that Oppenheim's original contention embodies a true conception of the disease.

The essential pathology is as follows : (1) persistence of various embryonic features (details given) in the nervous system ; (2) loss of, and changes in, anterior horn cells ; (3) absence of vascular and 'inflammatory' reactions ; (4) substitutive glial hyperplasia ; (5) fibre poverty in anterior roots and peripheral nerves ; (6) changes in muscle (fully described).

The problem for discussion may be expressed thus : Are the pathological alterations to be regarded as indicative of a process of degeneration in neural structures that have been normal ; or are they significant of an arrest of development, a quantitatively and qualitatively insufficient neuroblastic supply for a developing organism ? Bielschowsky examines this crux with great care and minuteness and decides for the latter hypothesis, viz., that the affection belongs to the class of congenital dysplasia or aplasia. Werdnig-Hoffmann's atrophy, on the other hand, is degenerative. He admits the occasional finding in myatonia cases of some evidence of degenerative change, and believes therefore that under certain circumstances more than one process may be responsible. But the general conclusion he considers thoroughly established on purely pathological grounds, and it finds support in the admitted clinical fact, that cases of amyotonia congenita sometimes exhibit definite and considerable improvement under observation, as has once more been shown in a recent case described by Bernheim-Karrer (*Zeits. f. Kinderheilk.*, xlv, 1928).

S. A. K. W.

[160] **The Czermak-v. Hippel disease of the retina** (Zur Kenntnis des Wesens der sogenannten Czermak-v. Hippelschen Netzhauterkrankung).—J. Meller and O. Marburg. *Zeits f. Augenheilk.*, 1928, lxvi, 1.

The condition described a number of years ago by von Hippel and Czermak has usually been considered an angiomatous formation of retinal vessels, but as long ago as 1913 this view was challenged by Meller, who believed it to consist in reality of a primary hyperplasia of glial elements in the same structure, and suggested the term 'gliosis retinæ diffusa telangiectoides.' The characteristic case here described in great detail was associated with a cystic glioma of the vermis and left cerebellar lobe, an association noted on numerous previous occasions and recently made the subject of study by Lindau.

A long critique of the views offered by Lindau can be summarised in the statement that pathological evidence proves the retinal condition to be primarily glial and not angiomatous, and the authors now propose it should be known as 'glioblastoma retinæ telangiectoides.' Numerous minutiæ of pathological interest are discussed in connexion with the case ; it is pointed out that

although gliomata are often considered to occur singly microscopical study sometimes reveals incipient glial nodules invisible to the naked eye. The multiplicity of the condition in cerebellum (or cord) and retina is not mysterious. Attention is directed to the absence of ' Xanthomzellen ' in the retinal tumour in this case, cells imagined by Lindau to be typical of angiomata.

S. A. K. W.

SENSORIMOTOR NEUROLOGY.

[161] **On the excitation processes of fits and other convulsive and emotional states.**—W. Burridge. *Jour. Ment. Sci.*, 1929, lxxv, 395.

The motor discharges of an epileptic fit are shown to possess a cardiac parallel. Examination of the cardiac parallel shows that the abnormal behaviour is due to excitation processes of too great intensity to permit the responding organ to give a normal response. Accordingly it is deduced that the responding organ, mediating mind, must also possess a limited capacity to give a normal response. Fits are thereby indicated to be based on excitation processes of too great intensity to evoke normal activity of the responding organ. The results lead to a consideration concerning which element of an excitation process, colloidal aggregation or electrolytes, holds prior possession of the field of response. Prior possession being conceded to colloidal aggregation, theories are developed of the mechanism of the process hitherto termed ' repression,' but better termed ' expulsion,' shell-shock and emotional states. Three kinds of unconsciousness are recognized: (1) That due to excitation processes of too small intensity. (2) That due to excitation processes of unfavourable composition. (3) That due to excitation of too great intensity.

C. S. R.

[162] **Some observations on experimentally produced convulsions.**—F. H. Pike, Charles A. Elsberg, W. S. McCulloch, and A. Rizzolo. *Amer. Jour. Psychiat.*, 1929, ix, 259.

Clonic convulsions arise from the motor cortex when this is anatomically and functionally intact. Tonic convulsions arise from lower motor mechanisms in the period immediately succeeding an injury to the cortical motor mechanisms. After lapse of sufficient time, clonic responses may be elicited from them. But this is not evidence that clonic convulsions may and do arise from lower motor mechanisms when those of the cortex are intact. Clonic convulsions in children may develop from lower motor mechanisms before the fibres of the pyramidal system have become myelinated and functionally active. All parts of the motor mechanism act together as one mechanism when the brain

is intact. It does not seem probable that one part should give rise to movements of one type, and some other part of the mechanism independently give rise to movements of another type, when the whole motor system is intact.

C. S. R.

[163] **Epilepsy in children : the relationship of water balance to the occurrence of seizures.**—IRVINE MCQUARRIE and CLARE H. HUSTED. *Amer. Jour. Dis. Child.*, 1929, xxxviii, 451.

THIS paper should attract attention from those interested in the biochemical problems associated with epilepsy. The production of ketosis by fasting on a suitable diet causes a marked loss in weight, the major portion being due to a loss of water. Several children with fits were accordingly observed while on different levels of water intake.

In the main four experiments were carried out. Firstly, in a boy suffering from frequent fits it was found that if fluids were forced fits became more frequent, while if fluids were restricted to the point of hyperthermia they ceased. Secondly, it was found that a borderline ketogenic diet along with rigid restriction of excess of fluids stopped fits and that if excess of body fluid was removed by the administration of urea a cessation of fits occurred. Thirdly, it was found that administration of the antidiuretic hormone of the pituitary resulted in the production of status epilepticus. Lastly a comparison was made between varying diets and different levels of water intake.

From the result of this work the authors appear amply justified in concluding that a disturbance in water balance is closely identified with the etiology of epilepsy. Many problems obviously arise as the result of this work and much research is necessary before any definite conclusions can be made ; but the work is certainly suggestive of a metabolic disorder being closely associated with epilepsy.

E. A. C.

[164] **Clinico-anatomical researches on the problem of syphilitic viruses** (Recherches anatomo-cliniques sur le problème des virus syphilitiques). —G. MARINESCO. *Ann. de dermatol. et de syph.*, 1929, x, 681.

THIS long paper embodies a complete historical survey of the controversies in respect of neurotropic and dermotrópic syphilitic viruses. Prof. Marinesco comes to the conclusion that so-called metasyphilitic cases, as is already well recognised, are not infrequently associated with lesions which belong pathologically to the dermotropic class, viz., vascularitis, miliary nodules, granulomas, etc. He does not accept the view that separate brands of spirochæte exist as far as neurosyphilis is concerned.

More interesting, because less appreciated at present, are considerations bearing on ' morbid predisposition ' to the affection. According to Friedmann,

79 per cent. of 100 cases of general paralysis between the ages of 24 and 50 belonged to the pycnic type of Kretschmer. The question of 'blood groups' has to be examined in respect of neurosyphilis. Experimental syphilis takes a malignant form in rabbits that have been thyroidectomised; and castrated animals of the same species develop the disease in a more general form than do the intact. The fact that in them nose, foot, and tail are the first parts to exhibit syphilitic lesions indicates a possible pituitary element. According to the author's investigations the pH of general paralytics tends towards alkalosis, being practically always above 7·40. A relationship exists between the severity of the lesions and alkalosis of the spinal fluid. In grave cases, with dementia, it reaches 7·58—7·59. In cases refractory to malaria it remains at a high figure. Lowering of pH forms a milieu unfavourable to spirochætal development. Using Ringer's solution and a particular variety of spirochæte he obtained the following results :—

In Ringer's solution	spirochætes are mobile during
pH=7·20	15 mins.— 2 hours.
pH=7·35	45 mins.—10 hours.
pH=7·50 }	72 hours.
pH=7·60 }	

If these experiments are corroborated they undoubtedly suggest that the terrain is at least as important as the organism, pathogenically considered: the degree of concentration of hydrogen ions in the cortex of the candidate for general paralysis is of significance for the appearance of that formidable affection. For cultural development there must be an optimum condition, which seems to exist in the cortex, especially in its middle layers.

S. A. K. W.

[165] **The cerebellar atrophies** (Études anatomo-cliniques sur les atrophies cérébelleuses).—P. Mathieu and I. Bertrand. *Revue neurol.*, 1929, i, 721.

This fine communication, for which the Prix Charcot was awarded in 1928, requires attentive consideration.

1. Phenomena of rigidity not unlike those of Parkinsonism are sometimes encountered in the course of olivo-ponto-cerebellar atrophies, in association with catatonia and dyskinesia. Analogous states of plastic rigidity are to be found in some instances of heredocerebellar ataxia. In no case were anatomical changes found in the basal ganglia, but in all the olivary system of the medulla was implicated. The interpretation of these clinical appearances is difficult; the suggestion is made that the cerebellum is one of the regulators of mesencephalic function, and that if its (cortical) inhibitory role is suspended tonigenic centres in the brainstem are set free, or its own ganglionic centres (dentate, roof nuclei), or perhaps both.

2. Certain cerebro-cerebellar atrophies pursue an acute or subacute course and are accompanied by disorders of the psychical series. They seem to belong to a special group of which examples have been published by other observers. A pathological basis in disseminated foci of necrosis associated with cortical atrophy is described. Most of the patients have been between the ages of 40 and 60.

3. The third chapter is concerned with familial cerebellar atrophies (Friedreich and heredo-ataxia cases). The former of these has mainly a dorsal systematisation in the cord, the latter an antero-ventral.

S. A. K. W.

[166] **Are epidemic encephalitis and Cruchet's encephalomyelitis identical? No.** (Sind die Encephalitis lethargica (epidemica) und die Encephalomyélite (von Cruchet) ein und dieselbe Erkrankung? Nein).—C. v. Economo. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1929, cxx, 265.

Most neurologists must be aware that Professor Cruchet of Bordeaux has recently republished in brochure form the remarkable series of observations made by him during the War on an epidemic of cases of encephalomyelitis (mainly in 1915–6–7) and has claimed that as a fact they were the first observations made on the condition now called epidemic or lethargic encephalitis; some indeed have gone so far as to rename this "Cruchet's disease." Professor Economo, than whom no one has a better claim to speak with authority, examines Professor Cruchet's arguments in a dispassionate way and concludes decisively against them. It would serve no useful purpose to summarise the details of the examination here, but the communication should be read in extenso because of the historical interest of the question.

S. A. K. W.

[167] **Remarks on the etiology of encephalitis after vaccination.**—D. Wiersma. *Acta Psychiat. et Neurol.*, 1929, iv, 75.

The author's observations may be given in his own words:

1. The clinical aspect, the histopathology and the epidemiology of encephalitis after vaccination and of other kinds of encephalitis which prove to be closely related to the former, make it impossible, or at least highly improbable, that the virulence of the vaccine, the bacteria or the albumen contained in it should be of importance in the etiology of any of these encephalitides, especially of that after vaccination.

2. Many arguments can be produced to support the hypothesis according to which the vaccine introduced into the human organism activates another virus already present in the body and causing, when activated in some way or other, the encephalitis.

3. The clinical peculiarities and the histological changes found in encephalitis after vaccination prove that this virus cannot be that of encephalitis lethargica.

4. It is possible, however, that pasteurellæ in this way take a part in the etiology of encephalitis after vaccination.

5. Perhaps a constitutional moment is of importance for its development.

M. C.

[168] **Writer's cramp in epidemic encephalitis** (Ueber Schreibkrampf bei Encephalitis epidemica).—Otto Sittig. *Klin. Woch.*, 1929, viii, 794.

A young man of 25, with characteristic writer's cramp, was found to have had epidemic encephalitis and to be suffering from moderate Parkinsonism. A few cases of this kind are on record; Sittig believes they should be regarded as instances of 'neurosis' for which an organic basis exists.

J. S. P.

[169] **The epidemic of disseminated neural inflammation in Poland during 1928** (Sur l'épidémie d'inflammation disseminée du système nerveux en Pologne durant l'année 1928; et sur ses rapports avec l'encéphalite léthargique, les atteintes du système nerveux au cours de certaines maladies infectieuses, et les cas aigus de sclérose en plaques).—Edward Flatau. *L'Encéphale*, 1929, xxiv, 619.

Prof. Flatau first describes some 17 cases of acute nervous disease observed by him in Poland during the earlier half of the year 1928. The general clinical picture is stated to have consisted of sudden onset of pains, paræsthesiæ, pareses or paralyses, sometimes including cranial nerves (usually the seventh), minimal sphincteric disorder, visual trouble, occasional convulsions and difficulty in speaking; brief hyperthermia or none; normal sleep. All the symptoms were moderate or slight, and commonly transient. Of the 17, only two proved fatal. In one case a spinal fluid pleocytosis of 112 lymphocytes with 80 neutrophils was obtained, and a ++ Nonne-Apelt reaction; in another, the latter finding, without increase of cells.

Pathological examination of one personal case gave the following: cellular infiltrates in basal ganglia, cord, and peripheral nerves; neuroglial reaction in the vicinity of vessels, with rare neuronophagia; infiltrates in leptomeninges.

A long but highly interesting discussion reveals the apparent trend in the author's mind; he would have it that recent epidemics (1924-8) of so-called disseminated encephalomyelitis are somehow the aftermath of epidemic encephalitis, which also is not unconnected with a seeming increase of mild or abortive cases of disseminated sclerosis in the same period. A useful bibliography is appended to the article.

S. A. K. W.

[170] **Study of the epidemic of infantile paralysis in Roumania during the year 1927** (Étude clinico-thérapeutique et anatomo-pathologique sur l'épidémie de paralysie infantile qui a sévi en Roumanie pendant l'année 1927).—G. MARINESCO and M. DRAGANESCO. *Ann. de l'instit. Pasteur*, 1929, xliii, 223.

THE 1927 Roumanian epidemic comprised some 1,576 cases, with a mortality of 10·02 per cent. In children of four years and under, the rate per cent. was 41·2 females and 58·8 males. For different age-periods it was 50 per cent. from 15 to 20 years, 33 per cent. from 6 to 9, 25 from 5 to 6, and 10 or under for most of the others. In Bucarest itself the rate over all was 9·5 (442 cases). Family or hospital contagion was remarkably low and in reality trifling; 202 children came from 56 families, and there were only 5 in which 2 children took ill at the same time. None of 80 children attached to the personnel of the hospital caught infection. But a house physician who had been treating cases for three months had occasion to visit an outlying village that had been completely immune, and one week later three cases developed there. He had given paper money to the villagers to buy cigarettes in an inn.

Treatment with convalescent serum did not, probably, have the best chance ; it was utilised in 19 cases, and was taken from children ill two months previously, under obviously difficult circumstances. Most of the 19 were already in the paralytic stage. Six were cured, 5 ameliorated, but with paralytic consequences, and 8 were fatal. With the serum of Pettit 30 cases were treated, and 6 ended in death ; 7 were cured, 6 had slight, 3 moderate, and 8 severe sequelæ.

S. A. K. W.

[171] **Diabetic tabes** (Zur Frage der diabetischen Tabes).—R. A. REYNOLDS. *Jahrb. f. Psychiat. u. Neurol.*, 1929, xlvi, 267.

THE author's case was that of a woman of 58, with well-marked diabetes mellitus, who also showed the ' tabetic ' symptoms and signs of sensory impairment in legs, loss of deep reflexes, Rombergism, etc. The pupils were fixed (both for light and accommodation). The Wassermann reaction in the serum was negative, but there seems unfortunately to be no record of the cerebro-spinal fluid.

Autopsy showed tabetiform alterations in the dorsal columns of the cord and in dorsal spinal roots. No histological sign of infection was present, and the diagnosis of pure degenerative change seems justifiable. According to the author lues is completely excluded.

J. S. P.

[172] **Systematised motor neuritis** (La neurite sistematizzata motrice).—O. Rossi. *Rassegna clin.-scientif.*, 1928, vi, 1.

A GENERAL discussion of the clinical features of so-called motor neuritis. Mention is made of the motor radial mononeuritis of lead palsy and its varieties and subdivisions; and of the motor polyneuritis of some cases of lead, malaria, and diphtheria. Other cases are etiologically obscure.

S. A. K. W.

[173] **The nervous disorders of erythræmia** (Zur Kenntnis der nervösen Störungen bei Erythræmie).—KEN TAGA. *Jahrb. f. Psychiat. u. Neurol.*, 1929, xlvi, 274.

A BRIEF series of references to previous work on the subject is followed by an account of the examination of a personal case, that of a woman of 72 (reds 9,100,000 at their maximum). The patient was also arteriosclerotic and suffered from cancer of the uterus.

In the meninges were found small hæmorrhages (chiefly pial), and in the brain diffuse œdema, with areas of cerebral softening of hæmorrhagic origin. The vessels in these parts were distinctly less affected than others in seemingly normal regions, which exhibited arteriosclerotic and endarteritic change. A status marmoratus was found in the basal ganglia. Papillœdema was present during life, but its causation was not determined.

S. A. K. W.

PROGNOSIS AND TREATMENT.

[174] **Cerebral arteriography and cranial hypertension** (L'artériographie cérébrale et l'hypertension cranienne).—E. MONIZ. *Revue neurol.*, 1929, i, 1122.

[174] **Three new cases of cure of cranial hypertension by intracarotid injections of sodium iodide** (Trois nouveaux cas de cure, au moins provisoire, du syndrome d'hypertension cranienne par les injections intracarotidiennes d'iodure de sodium).—E. MONIZ. *Revue neurol.*, 1929, i, 1135.

IN these two papers the author reports cases in which pronounced amelioration of the general symptoms of rise of intracranial pressure took place in consequence of intracarotic injections of sodium iodide for purposes of arteriography. Headache disappeared, and in one or two instances improvement in vision unmistakably occurred, though transient. It is especially in cases of serous meningitis that such change for the better is remarked and it may persist indefinitely. He believes it possible to diagnose the latter condition arteriographically when the arteries of the Sylvian group are not visible in the first part of their course; on the other hand the condition may occur in regions not, as a rule, capable of examination by this particular technique.

J. V.

[175] **Arterial encephalography in cases of cerebral tumour** (L'épreuve de l'encéphalographie artérielle dans le diagnostic de quatre cas de tumeurs cérébrales operées).—E. Moniz, A. Pinto, and A. Lima. *Presse méd.*, 1929, April 17, no. 31.

Continuing his researches on arterial encephalography by the technique previously described (see this Journal, vol. ix, p. 247), Moniz and his collaborators report four cases of cerebral tumour successfully operated on, in which series two were without any localising symptom or sign. Study of the arteriogram proved to be of direct service in localisation. The value of the method in selected cases seems undeniable.

J. V.

Endocrinology.

[176] **The relation of endocrinopathic states to conduct disorders of children.**—Louis A. Lurie. *Amer. Jour. Psychiat.*, 1929, ix, 285.

The endocrine glands are the dominant factors in determining bodily structure. Psychiatrists should bear in mind that the correlation between structure and function long noted by physiologists includes both mental and physiological functioning. Disturbances in glandular function may lead to abnormal conduct reactions either directly or indirectly: directly, as in cretinism, where the lack of thyroid secretion is responsible for the mental retardation; indirectly, as in those cases in which as a result of physical malformation, such as midgets and giants, the individual develops abnormal mental attitudes and personality traits which make normal social adjustments very difficult if not entirely impossible. The close relationship existing between endocrine disturbances and delinquency, incorrigibility, sexual perversion, crime and other antisocial states has been demonstrated in a sufficient number of cases to warrant the statement that no study of a child who presents a behaviour difficulty is complete without a thorough study of the glands of internal secretion.

C. S. R.

[177] **Suprarenal virilism with a report of two cases.**—A. Dingwall Fordyce and Howell Evans. *Quart. Jour. of Med.*, 1929, xxii, 557.

The two cases reported are typical of the condition.

It is of interest that both had an extreme redness of hair and that one case had a deformityof the larynx comparable to that found in cases of congenital laryngeal stridor.

E. A. C.

[178] **Thyroneural dystrophy: the association of congenital myxoedema with mental and neuromuscular disorders.**—Walter M. Kraus, Samuel Brock, and Paul Sloane. *Amer. Jour. Med. Sci.*, 1929, clxxviii, 548.

The authors describe two cases of this malady occurring in one family. In one, cretinism was early observed: at school the patient was backward and at the age of 21 developed a peculiar gait. When she attempted to speak she stuttered and there were marked associated movements of the face and upper limbs: there were flexor movements of the trunk and hips; extensor plantar responses were present, while the feet showed a typical pes cavus. In the other case, also that of a cretin, the feet were reminiscent of pes cavus and the plantar responses were sometimes extensor in type.

They have abstracted the literature on the subject and show that nothing specific is known as to the pathology of the disease.

E. A. C.

[179] **The mongol: a new explanation.**—R. M. Clark. *Jour. Ment. Sci.*, 1929, lxxv, 261.

The writer's view that mongolism is caused by fœtal hyperthyroidism ceasing at birth is based on the theory that if the known actions of hyperthyroidism on the embryos of animals were at work on the human fœtus, the characteristic features of the mongol would be produced. Fœtal hyperthyroidism could not fail to cause abnormal endocrine inter-reaction, and it has been said that in every mongol some endocrine disturbance can be demonstrated. The general arrest of growth and development, including that of the skull and brain, can be thus explained. The coincident congenital anomalies could be similarly accounted for. If the retarding actions of thyroid on growth and development affected a part of the embryo when it was recapitulating a pre-human stage of its evolution, it is conceivable that resemblances to pre-human features might result, which would explain the so-called 'reversions.' Hyperthyroidism and the mechanics of exophthalmos, with Moebius's sign, occurring during antenatal life and ceasing at birth, would account for the changes in and around the eyes of the mongol. The usual explanations given to account for the characteristic tongue are not in conflict with a hyperthyroidic theory, but in Clark's view the mongol's tongue results from participation of the tongue muscles in the general muscular hypotonus. The incidence of mongolism in twins suggests the possibility that the placenta may be at fault, and it would be interesting to determine what effect feeding thyroid to pregnant mammals would have on the offspring.

C. S. R.

Psychopathology.

PSYCHOLOGY.

[180] **Tests of temperament and personality.**—P. E. VERNON. *Brit. Jour. of Psychol.*, 1929, xx, 97.

TEMPERAMENT and personality are of extreme social, educational and industrial importance, so that it is very necessary to devise satisfactory methods of diagnosing, estimating and measuring them, and of establishing more clearly their psychological nature. The rating method is scarcely practicable, although preferable to the ordinary personal interview. In general, objective tests neglect the complexity of the subjective factors involved; owing to their very objectivity and scientific orthodoxy they are artificial and psychologically unsound. Personality and temperament are organized and integrated wholes which cannot be put into simple quantitative terms; their estimation must be supplemented by direct qualitative observation and intuition. Another approach intermediate between the objective test and the subjective interview, as used by Köhler and Burt, was tried with far more promising results. Various simple performance tests and games were given to a number of subjects under conditions that were as normal as possible. Their remarks and behaviour were observed and many traits emerged. Chief of these were æsthetic and scientific constructive interest, planning capacity, emotional instability and variability, extraversion-introversion, suggestibility and persistence. They appear to agree well with the subject's characteristics in real life, and the method is capable of considerable development, systematization and practical application.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[181] **A review of visceroptosis and allied abdominal conditions associated with invalidism.**—H. BEDINGFIELD. *Quart. Jour. of Med.*, 1929, xxii, 611; 1929, xxiii, 1.

AFTER reviewing the literature on the subject, in all 641 references, the author reaches the following conclusions:

1. The position of the viscera within the abdomen may vary to a considerable extent and yet be well within the range of normality.

2. There is no necessary relationship between the body build of the individual and the position of the abdominal viscera beyond that imparted by mechanical necessity. That is to say, ceteris paribus, the organs will be higher in a short broad abdomen than they will in a long narrow one.

3. There is a considerable range of movement of the hollow viscera which is normal for any given individual, so that their actual position in health may vary from time to time.

4. This variation in position may be brought about by either physiological or psychical factors.

5. The position of the viscera, per se, plays no part in the production of symptoms. It only does so in rare cases when associated with a true abnormality, as for instance an aberrant renal artery.

6. While one or several of the symptoms discussed may appear as transient phenomena in any individual, their persistence is only met with in those of a special constitution.

7. This constitution is not associated with a special body build, but rather with a state of nutrition and state of mind.

8. This state of nutrition betrays itself in the tall thin individual by underweight, and in the short broad individual by overweight. In both it is accompanied by poor muscle tone leading to postural defects. The state of mind reveals itself by abnormal mental reactions.

9. The association of malnutrition, poor muscle tone, and abnormal mental reactivity is characteristic for this group of cases.

10. This association appears to depend on a congenital, possibly inherited inability of the individual to adapt himself satisfactorily to the various strains and stresses of life.

11. One of the failures of adaptation may be lack of resistance to degrees of infection and toxæmia which have no effect on normal individuals. This may account for the failure to discover the 'toxin' present in cases of autointoxication.

12. When sensations from malfunctioning viscera rise into consciousness in an individual who is the subject of repressions based upon an inferiority-complex, these tend to be utilized in the form of symptoms to reinforce the repression. The malfunction may originate peripherally from local causes as improper feeding, etc., or centrally from weakening of inhibition produced by fresh mental conflicts.

13. Later, with the establishment of a vicious circle of malfunction-symptoms-malfunction, actual tissue changes may occur in the affected organs, rendering them incapable of a return to normal function.

14. When this occurs there is little chance of restoring the individual to his or her particular standard of health.

15. The treatment of the condition is essentially preventive.

E. A. C.

PSYCHOSES.

[182] **A group of benign chronic psychoses: prolonged manic excitements. With a statistical study of age, duration and frequency in 2,000 manic attacks.**—F. I. WERTHAM. *Amer. Jour. Psychiat.*, 1929, ix, 17.

A HISTORICAL survey of the literature bearing on prolonged manic excitements is given. Fully described cases are scarce and there is little agreement as to

the nature, outcome, and clinical evaluation of these cases. The view seems to prevail that there are states of 'chronic mania' which are essentially incurable, while others really belong to the schizophrenic group. Six cases of from five to eleven years' duration are reported. Four had other manic attacks: in two the prolonged excitement was the only attack; no patient had depressive phases before or afterwards. One patient had two attacks of prolonged manic excitement, each of six years' duration. Three patients recovered; two died during the attack; one is still affected. The three recovered cases had later manic attacks. The onset of the prolonged attack occurs in mature or advanced age. The premorbid personality of the six cases shows a "positive charge in mood and activity"; they are very active, persistent, talk a great deal, have many friends and an optimistic nature. All cases were free from "growth disorders" and three belonged definitely to the pyknic type. After years of psychotic behaviour the integrity of the personality was well preserved and there was little or no diminution of intellectual discernment. Conditions favourable to the occurrence of prolonged manic attacks are: (*a*) the existence of a manic constitution combined with what may be termed (*b*) an increased "psychobiological rigidity."

There are evidently different groups of psychoses with prolonged manic excitements. The group isolated and here described as cases of benign chronic psychoses has distinct constitutional and clinical features and may serve as a point of orientation for further investigation of prolonged excitements in affective psychoses and of chronic excitements in general. Practically, its recognition is of importance because these cases are frequently wrongly interpreted. On account of the long duration, the intense outbursts on paranoid lines, the great impulsiveness and the contrary attitude, the cases are apt to be diagnosed as paranoid psychoses, as schizophrenia, as paraphrenia, or as involutional psychoses of unfavourable type.

It seems possible to single out a distinct group of benign chronic manic excitements which consists of infrequent cases arising on a foundation of a manic constitution with a tendency to recurring acute manic attacks, an affinity to the pyknic body type, a tendency to the onset of a chronic condition in mature or later life, absence of depressive attacks or circular phenomena, reduction in the manic overactivity with tendency to fluctuation, absence of pronounced signs of deterioration, and a potential possibility of recovery from the chronic attack.

C. S. R.

[183] **A study of the functional psychoses in childhood.**—Jacob Kasanin and Moses Ralph Kaufman. *Amer. Jour. Psychiat.*, 1929, ix, 307.

Psychoses in children are relatively rare. There were only 160 children under the age of 16 of a total number of about 6,000 patients who were admitted to the Boston Psychopathic Hospital in the years 1923, 1924, 1925. Only

65 children were found to be psychotic. Twenty-one of these were diagnosed as dementia præcox and four as manic-depressive insanity. These two groups were studied and re-classified. The children diagnosed as manic-depressive psychotics did not necessitate any change in diagnosis. The 21 cases diagnosed as dementia præcox were grouped as follows: six were considered to be typical cases of schizophrenia; five had reactive psychoses; two were regarded as cases of 'Propfhebephrenie'; two showed evidences of a toxic psychosis; two cases exhibited essentially hysterical reactions; one case had a traumatic psychosis; two cases were essentially atypical or psychopathic personalities and one case remained undifferentiated. The outcome was as follows: two children died, seven are still patients in state hospitals, and 16 are back in the community. The toxic and the typical schizophrenic cases are the most ominous. All the toxic cases died. Of the six typical schizophrenic cases and the two cases of 'Propfhebephrenie' only three patients were able to go back to the community. Physical factors, including menstruation, were of significance only in a few cases. Heredity was definitely poor in the clear-cut cases of schizophrenia and the affective disorders. Poor heredity was also present in some of the other cases. Sex with its various problems played an important rôle in most. Environmental stress in its various forms seems to be of definite etiological significance. The content of the psychoses gives only limited knowledge of the prepsychotic problems of the children, except in a few cases.

C. S. R.

[184] **Significance of narcissism in the psychoses.**—Mary O'Malley. *Psychoanalytic Review*, 1929, xvi, 241.

While narcissism is a normal, constructive force in the personality, an excess or decrease of this element is a pathological expression, a compensatory inferiority reaction, or one of libido-fixation at lower integrated levels. In the psychoanalytic treatment of these cases, the following points must be observed: The same methods may not be applicable to the analysis of psychoneurotic and frankly psychotic patients. In treating the former it is found that there has been a faulty construction of the ego-ideal, but generally the patient, with the assistance of the analyst, has sufficient forces within himself to build up an ego-ideal. Under these conditions the duty of the analyst is to bring forth repressed material and reconstruct the personality including the ego-ideal, in accordance with a reality principle. In the patient with a psychopathic personality there is a weak ego-ideal. This maladjusted class of patients cannot be reached by psychoanalysis because of excessive self-love and inability to transfer libido to a love-object or to accept any principle consonant with social well-being. In the psychotic patient there has been an extensive injury to, and sometimes destruction of, the ego-ideal, with

consequent regression. Narcissistic forces have been hampered or destroyed and the patient does not always have the capacity to restore or rebuild the ego-ideal: he requires assistance in this direction. A reconstruction of the personality in these cases takes place by means of an educational process similar to that given to children. In the psychotic patient many of the repressions have risen into consciousness, producing abnormal behaviour symptoms, and in treatment the instinctive forces must be recognized, dealt with, and directed into socially acceptable channels. This involves analysis of the ego-ideal. Psychoanalytic success with psychotic patients depends on the degree to which they can be made to understand the real motives behind their symptoms and can be brought consciously to correct their behaviour in the direction of socially acceptable standards.

C. S. R.

PSYCHOPATHOLOGY.

[185] **A note on urinary reactions in relation to intestinal toxaemia in psychotic subjects.**—S. A. Mann and H. L. Shipp. *Jour. Ment. Sci.*, 1929, lxxv, 420.

In common with other investigators these writers have found that the Buscaino black reaction in the urine is associated merely with the concentration of normal urinary constituents, and conclusions based on the incidence of this reaction in the urine of psychotic patients in regard to the existence of toxic substances or the possible influence of intestinal toxæmia are fallacious. The same applies to the value of crude tests on untreated urines, such as the simple addition of Millon reagent. The technique attached to such investigations presents extreme difficulty and the cases under examination should be rigorously controlled as regards diet and time of collection of specimens. No simple tests of the urine are of any value and all investigations should be on the scheme elaborated by Stewart, with chemical and numerous normal controls. The evidence so far available does not warrant the assumption, based on urinary reactions, that the presence of bodies arising from protein putrefaction has any peculiar significance in psychotic subjects. If such be the case it is necessary to assume the presence of some hitherto undiscovered toxic substance arising from intestinal toxæmia, or to assume that the psychotic organism is more prone to be affected by putrefactive products than is the normal organism.

C. S. R.

[186] **The Buscaino black reaction in urine. Its genesis and diagnostic value with special reference to mental diseases.**—S. Katzenelbogen. *Amer. Jour. Psychiat.*, 1929, viii, 1021.

This reaction was studied chiefly in psychoses and (for purposes of control) in diseases of other systems and in normal individuals. In the group of nervous

and mental diseases, of the total number of 147 patients, 43, or 29·25 per cent., showed a positive reaction. The latter was most frequently positive in 'anergastic' disorders (organic reaction types) viz., in 47.5 per cent. of the 40 cases. In the groups of 'thymergastic' (manic-depressive), 'merergastic' (psychoneuroses), 'parergastic' reaction types (schizophrenia, paranoia), epilepsy and some unclassified cases, it was positive in a percentage between 11·1 and 33·3. In the control cases a positive reaction was obtained in 42·5 per cent. of the 40 cases of acute and chronic affections of different systems, and in 8·6 per cent. in the group of 35 normal individuals. In the specimens of urines with positive black reaction the ratio between uric and sodium chloride was higher than in the urines with negative reaction. Both uric acid and sodium chloride were found in lower amounts in the positive cases than in the negative ones; and the high ratio between these two chemical compounds in the urines with black deposit is essentially due to a low concentration of sodium chloride. In the light of the fact that, on the one hand the black reaction was found positive in varying pathological conditions and even in a few normal individuals, and on the other hand that the positive reaction was accompanied in most cases by a higher ratio between uric acid and sodium chloride than the negative reaction, the conclusion is justified that the Buscaino reaction does not depend upon the diseases themselves, but only upon epiphenomena. These changes are probably, at least partly if not altogether, due to the diet of hospital patients, which is usually reduced in proteins and salt. The findings are thus completely unfavourable as to the value of the reaction.

C. S. R.

[187] **Comparison of Kahn and Wasserman tests in 368 mental patients.**—F. Proescher. *Amer. Jour. Psychiat.*, 1929, ix, 195.

Throughout there was a close correlation of the two tests. In two cases of dementia præcox (acute stage) positive Kahn tests were observed with persistingly negative complement fixations and without clinical evidence of syphilis. After about one month each gave negative reactions with each test and without any treatment. A false positive reaction may occur, due to an unknown change in the colloidal stability of the serums or a congenital syphilis, since false negatives in the latter condition are also observed. Besides dementia præcox, false indications were observed in two cases of alcoholism without clinical evidence of syphilis though the actual presence of the disease could not be ruled out with certainty. Both Kahn and Wasserman reactions were positive on the patients' admission. Subsidence of the acute symptoms in each case brought negative results in both serological tests. These cases were followed for several months without further positive findings. Complete disagreement was noted in one case of senile dementia, in arteriosclerosis, manic-depressive psychosis, involution psychosis, toxic psychosis, narcotic

disorder, and cerebrospinal syphilis. In general paralysis there were 11 disagreements in 66 cases—a high percentage. In some treated cases the Kahn test remains positive longer than the complement fixation. The few exceptional cases noted may suggest that false serological reactions may occur with mental or nervous conditions which clear up without further treatment than some few weeks of rest. The question awaits more evidence.

C. S. R.

[188] **Dermographia in the insane.**—A. W. H. Smith. *Jour. Ment. Sci.*, 1929, lxxv, 458.

Dermographia in the insane tends to occur slightly more frequently among females than males, and with less consistency with regard to the related phenomena of urticaria. Dementia is the only class of insanity which shows a percentage of dermographia sufficiently in excess of the average to warrant its being claimed as a characteristic phenomenon, but the total percentage is even then so small as to preclude any suggestion of its being pathognomonic. While dementia præcox is said to be the commonest form of insanity to show dermographia, the most conspicuous examples are to be found in other classes, so that in any individual case the symptom cannot be regarded as diagnostic in any more than a relative sense and of proportional valuation.

C. S. R.

[189] **Tuberculosis from the psychiatric approach.**—Anita M. Muhl. *Psychoanalytic Review*, 1929, xvi, 397.

Since the majority of people are infected with tuberculosis at some time and only comparatively few of them break down with a chronic form in adult life, there must be some factor other than the tubercle bacillus which is responsible for the failure of the body to defend itself against the disease. In view of present-day knowledge with regard to colloid structure, the mechanism of the vegetative system, and the fact that repressed unhealthy emotions, though unrecognized by the subject, act through the sympathetic, causing a rise of the metabolic rate with attendant toxic symptoms and destructive de-energizing structure changes, submerged emotional conflicts can be regarded as an important phase in the lowering of resistance. Certain unfavourable traits and trends may appear in any human being, but in the individual who breaks down with chronic tuberculosis in adult life they become exaggerated; of these traits and trends a definite group is constant for the respiratory type which develops chronic tuberculosis. The common features for the adults are inertia, fatigability, oscillating mood, perseverations, irritability, masochistic and sadistic traits, suggestibility, hypersensitiveness, selfishness, regressive and suicidal trends, depression and abnormal respiratory behaviour. Other frequent characteristics for the adults are ambition, evidence of dissociative

trends, memory impairment and day-dreaming. The regressive feature is deeply rooted and the so-called optimism is only a compensatory reaction for the depression and suicidal trends which have been found in the unconscious of all the patients. So-called 'pathological optimism' is not present in the child with incipient tuberculosis but the tendency toward depression is present. Common characteristics in children are: suggestibility, irritability, depression, fear, anxiety, selfishness and intensified type expression (energy imbalance). If children with incipient tuberculosis can be re-educated emotionally and have their energies directed along the right lines so that they would be free from repressed emotional conflicts it is argued that there would be little adult chronic tuberculosis. It is held that psychotherapy as a means of releasing these pent-up conflicts and of helping to readjust these patients, adult as well as juvenile, has a distinct place in the treatment of tuberculosis.

C. S. R.

PROGNOSIS AND TREATMENT.

[190] **Notes on the emotion of fear as observed in conditions of warfare.**—F. Aveling. *Brit. Jour. of Psychol.*, 1929, xx, 137.

After dealing with the nature of fear, its antecedents, its immediate and after-effects, its control is discussed. The chief methods indicated are:—(1) Drugs, alcohol, etc., which have a more or less immediate beneficial effect but later are favourable to the genesis of fear. (2) Bodily activity: this is probably due to more than one factor. There is the draining off of nervous energy through the nerves supplying the muscles involved, and also the distraction of attention. (3) Breathing, deep and regular: this has its repercussion on the circulatory and perhaps other systems, thus tending to counteract some of the unusual and irregular physiological activities which increase the emotion. (4) Singing, especially chorus singing aloud; the rhythm makes for good breathing, attention is distracted, and through a sense of gregarious solidarity inhibitions to fear are set up. Probably also the tune and words of the song arouse associations which drain off energy from the emotion. (5) Distraction of attention would seem to be one of the most efficacious methods of controlling fear. The desired effect may be brought about in various indirect ways, thus providing a motive for self-control. (6) Feeling or sense of responsibility: this would apparently involve the mechanism of distracted attention, at the same time providing motive for self-control. (7) Human respect: shame of appearing to be afraid; possibly also thoughts or representations of the penalties of giving way to fear and cowardice. (8) Tranquil conscience: this may be secured chiefly by religious and moral means. (9) Imagination and instinctive false judgment: the practical instinct to take cover manifests itself in greater confidence and decrease of fear even when cover is totally inadequate and worse than useless. (10) Emotional conflict:

it is possible to decrease, and even totally to inhibit fear by securing its conflict with other emotions such as anger or hatred. At the same time it must be noted that fear and anger, at any rate in their more feeble forms, can coexist simultaneously in consciousness. (11) Suggestion: predisposition to fear attacks is somewhat lessened by the use of suggestions given in the hypnoidal state. Simple suggestion during a fear crisis is often distinctly beneficial. A superstitious belief in mascots and charms acts as a species of suggestion with appreciable results. (12) With regard to the removal of mental and physiological effects on the symptoms of fear (shock) which persist when the actual emotion is removed, these have been treated with remarkably good results by simple suggestion, suggestion with faradization, hypnoidal suggestion and hypnosis.

C. S. R.

[191] **Twenty cases of general paralysis of the insane treated by benign tertian malaria.**—JAMES H. MURDOCH. *Jour. Ment. Sci.*, 1929, lxxv, 471.

THE results obtained were: Cases discharged 10; greatly improved, 2; arrested, 1; progressed, 2; fatal, 5; cerebrospinal fluid improved, 12. It would appear to be a difficult matter to decide when it ceases to be worth while giving the treatment. A patient who was discharged had been insane for two and a half years and had had congestive seizures; another who improved greatly had lost control of the sphincters and was bedridden. All the deaths occurred in advanced cases which were bedridden before treatment. The most marked change in the cerebrospinal fluid was in the cell count, which came down to 5 at most in those cases which showed marked improvement. The globulin content was diminished slightly, but the Wassermann reaction and the colloidal gold curve remained unchanged. Of the patients who were discharged, one died six months after going home but no physical signs of general paralysis were present. It is now three years since the first of those cases was treated and more than two and a half years since the first was discharged. All of the discharges have been at home at least a year, and all of them are following their usual occupations. These remissions are of considerably longer duration than natural remissions, and it seems reasonable to suppose that they would not have occurred had the patients not received malaria therapy.

C. S. R.

[192] **Dementia praecox. A trial and a retrospect.**—HENRY J. BERKLEY. *Amer. Jour. Psychiat.*, 1929, viii, 1085.

LAMENTING the lack of progress in the treatment of dementia præcox, the writer was led to try the effects of typhoid-paratyphoid vaccines, since they favourably influence the course of cerebrospinal syphilis. Eight subjects were chosen; four white males, two white females, and two male negroes, with

ages from 17 to 38 years, the last representing the longer deteriorated types. One cubic centimetre of the sterilized vaccine selected represented two billion germs. The initial dose was one-tenth of 1 c.c., increased by degrees to .15 c.c. according to the reaction produced, and the vaccine was injected, intravenously, every other day for sixteen days. The average rise in temperature was to 102.4°. Four of the cases showed no improvement; two seemed somewhat improved but the change was not definite; and in two others the disease was aggravated. Thus, the author concludes, another experiment has ended unfavourably.

C. S. R.

[193] **Light therapy in mental hospitals.**—H. Dove Cormac. *Jour. Ment. Sci.*, 1929, lxxv, 410.

Actinotherapy has been practiced at Parkside Mental Hospital for nearly four years, and from the writer's experience is a very necessary adjunct to the treatment of mental cases. It is here employed as a routine for convalescent cases, whose recovery is thereby hastened. Care is needed in the choice of cases for treatment. Mention should be made of the excellent results obtained in dementia præcox of the simple type, and also in the hebephrenic after the acute phase has passed. In the author's opinion every mental hospital should be equipped with ultraviolet-ray apparatus for the treatment of mental disorders, as well as for the many physical diseases which are ameliorated by its use.

C. S. R.

Reviews and Notices of Books.

Principles of Experimental Psychology. By HENRI PIÉRON. Translated by J. B. MINER, Ph.D. London: Kegan Paul, Trench, Trubner & Co., Ltd. 1929. Price 10s. 6d.

THE title of this work is slightly misleading. It deals less with principles and much more with results. The latter are well arranged, but are stated in a cursory way with few references and sources, from the labour of providing which the author expressly excuses himself. Part one, on the reaction processes and the forms of behaviour, and part two, on the affective reactions, are disappointing on account of their vagueness. The traditional difficulty in distinguishing reflexes and instincts is quickly surmounted by regarding them as extremes of a continuous series. After stating—what is quite true—that the terminology of the domain of affect is extraordinarily inexact and variable, the author does not clarify the situation. He says "The agreeable and disagreeable are designated by very different names: sensation, sentiment, emotion and affection. As all these words are used in other meanings, we shall say that these processes are elementary affects. Pleasure and pain are emotional forms of the elementary affects. Emotion is essentially affective reaction of an intense character." The reader is none the wiser. It may be the translation (we have not been supplied with an original) that is at fault, but such a paragraph as the third on page 59 hardly seems to make sense as it stands.

The bodily manifestations of emotion are simply the spilling-over of psychic energy—mere by-products: the criterion of biological usefulness is hardly considered. Memory and motor habits are identified with each other. The experimental work on memory is considered to give results strictly homologous with Pavlov's on the acquisition of conditioned reflexes.

The whole book is well arranged, but gives the impression of having been rather hurriedly written.

Problems of Neurosis: a Book of Case-histories. By ALFRED ADLER. With a Prefatory Essay by F. G. CRUICKSHANK, M.D., F.R.C.P. Edited by PHILIPPE MAIRET. London: Kegan Paul. 1929. Pp. 178. Price 8s. 6d. net.

DR. ADLER'S new book on the problems of neurosis is well worth pondering over in a quiet hour, since its pages provide the reader with a series of intimate glimpses of clinical histories—fragments from life of maladjustments, misfits, efforts, struggles, and compensations. The types of case here narrated are

precisely those with which every practising physician comes in contact, where the patient needs guidance and direction much more than any specific treatment. One admires both the skill in analysis and the human sympathy revealed by the author as he turns over the sheets of his case-book. Medical and psychiatric technique is discussed as required, but the major outlines of the book consist of explanations of the genesis of symptoms and of the sequels to inferiority situations. The simplicity with which these are set forth conceals to some extent the consummate knowledge of human nature behind it.

Dr. Cruickshank's preface forms a mise-au-point for the reader who wishes information in regard to the present-day status of Adler's individual psychology.

Science and Personality. By WILLIAM BROWN, M.A., M.D., D.Sc. With a foreword by Sir OLIVER LODGE. London: Oxford University Press; Humphrey Milford. 1929. Price 12s. 6d. net.

THE nucleus of this book consists of several essays and addresses published elsewhere by Dr. Brown, supplemented by chapters containing elementary accounts of various aspects of psychology, psychopathology, and religion; by an appendix on a spiritualistic séance, a preface by Sir Oliver Lodge in which the author's qualifications for the catholic task he has imposed on himself in this book are set forth, and by a letter from a grateful patient describing some of Dr. Brown's methods from the patient's point of view. The writing is clear and the book should appeal very much to those for whom it is intended. Biological science is accepted as more fundamental than physics and chemistry on the basis of its organic preoccupation. God is the most concrete reality and an acceptance of the Christian religion is assumed. God is the concretisation of all values; values are such things as satisfy individual needs; values grow with the organisation of sentiments. Ultimate values are referred to, but not elucidated. With these views, the author makes a rather half-hearted essay at reconciling analytic psychotherapy with spiritual healing. The psychoanalytic views of the origin of religious belief are discarded as crude, but nothing very definite is substituted. Dr. Brown performs a useful service in emphasising the importance of the intellect as a factor of instinctive force, and by calling attention to the unwarrantedly complete depreciation of intellectual constructions by Freudian theory.

Handbuch der Psychiatrie. Edited by Professor Dr. G. ASCHAFFENBURG. Allg. Teil: Bd. II. Spezielle Histopathologie des Grosshirns. By Prof. Dr. A. JAKOB, Hamburg. Leipzig and Vienna: Franz Deuticke. 1929. Pp. 459–870. Price Mk. 40.

WE have already reviewed (this JOURNAL, vol. viii, p. 187) the first part of Prof. Jakob's treatise on cerebral neuropathology, enumerating its noteworthy features and praising its conspicuous merits. The companion volume dealing with the special histopathology of the brain fully maintains the wonderfully high standard of its predecessor; some 266 illustrations, many of which are

coloured, constitute in themselves a kind of atlas which is sure to prove useful. The subjects dealt with comprise tuberculosis of brain and meninges, syphilis of the same, trypanosomiasis, disseminated sclerosis, and diffuse sclerosis. Nearly 200 pages are reserved for the pathology of general paralysis—an impressive example of the elaborate scale on which the work is planned. One of the particularly interesting sections discusses syphilitic arteritis of the small cortical vessels, an important subject to which little or no attention has been given in England. We note also the attraction of the pages concerned with the debatable question of tabetic psychoses distinct from those of the more serious affection. The problems of disseminated sclerosis are examined rather shortly, and their bearing on disseminated encephalomyelitis investigated; the author cites with approval the view expressed by Wohlwill, that when there is talk of the latter passing into or blending with the former it is not the respective diseases, but our conceptions of them, that thus seem to mingle.

This work is much the finest modern textbook of neuropathology with which we are acquainted and as a work of reference may fairly be considered indispensable.

Handbuch der Psychiatrie. Edited by Prof. Dr. G. ASCHAFFENBURG. Spez. Teil: Bd. I, Teil 2. Die Optisch-Agnostischen Störungen. By Prof. Dr. OTTO PÖTZL, Prag. Leipzig and Vienna: Franz Deuticke. 1928. Pp. 358. Price Mk. 25.

THE different varieties of so-called mind-blindness are divided by Prof. Pötzl into three chief groups: the first consists of visual agnosia in respect of apperception as a whole, or failure to recognise objects by sight. The second includes more special or limited subvarieties, such as pure word-blindness, congenital word-blindness, failure of colour perception ('optic aphasia' for colours), figure- and music-blindness, and others. In the third are placed still other peculiarities such as cerebral metamorphopsia, central diplopia, defective visual orientation, the 'visual-mind-paralysis' of Balint (inability to look in certain directions without visual field defect or peripheral motor lesion), etc. A fourth section deals with certain complex conditions such as derangement of distance appreciation, loss of faculty of forming visual images, and forms of visual agnosia due to cerebral trauma.

As a compendium of what is known of a complex and difficult subject, Dr. Pötzl's monograph deserves careful perusal. We have merely summarised some of its features sufficiently to indicate its width of conception, and cannot here enter on any detailed commentary. It may be noted, however, that dysmegalopsia of central origin, and some analogous disturbances associated with hysteria, epilepsy, and other states, do not seem to have attracted the author's attention, although calculated to throw light on an obscure problem.

Les Troubles Mentaux de la Sclérose en Plaques. By Dr. ANDRÉ OMBREDANE, Paris. Paris: Les Presses Universitaires de France. 1929. Pp. 352. Price not stated.

IT is satisfactory to find increasing attention devoted to what may be termed the organic approach to mental problems. Of this Dr. Ombredane's work provides a conspicuous example. He has undertaken an analysis of the 'mental' symptoms of disseminated sclerosis, and substantiates views expressed by Cottrell and Wilson to the effect that they are far more frequent than has hitherto been allowed. No fewer than 47 of 50 cases exhibited such phenomena, though the exact sense of the word 'mental' is not defined, or, rather, is taken very comprehensively. Three classes of symptom can occur, viz., disorders of affectivity and intellect (the 'polysclerotic mental state'), conditions of dementia, and psychoses. The author finds 'intellectual' impairment almost as common as affective, including therein difficulty in sustained mental effort, reflective fatigability, imperfection of memory, slowness in comprehension and poor mental analysis and synthesis. Only in a small minority are demented and psychotic states recognisable: mental confusion is rare, though anxiety is declared to be a fundamental featrue of the disease. From the viewpoint of pathogenesis a theory is advocated which ascribes such 'mental' symptoms less to the presence of cortical and subcortical plaques of sclerosis than to the existence of a diffuse toxi-infective process.

A good bibliography is appended, and a large number of personally observed illustrative cases.

Handbuch der Neurologie des Ohres. Edited by Prof. Dr. G. ALEXANDER, Prof. Dr. O. MARBURG and Dr. H. BRUNNER. II Band, 2 Teil. With 242 illustrations, some in colour. Berlin and Vienna: Urban & Schwarzenberg. 1929. Pp. 723–1950. Price Mk. 150.

WITH this volume the fine textbook of aural neurology compiled by members of the Austrian and other continental schools of medicine is completed. Some 1,200 pages long, the new part is superbly illustrated and printed in a readable type. Among its more important contributions from the wider viewpoint of the neurologist (as compared with the otologist) are those devoted to intracranial complications of inflammatory ear disease, and to involvement of the eighth nerve in various general toxic conditions. Long articles by two of the editors cover the field of otogenic temporal and cerebellar abscess respectively; and nearly 300 pages are required for a discussion by Prof. Bonvicini of aphasic disorders attributable to temporal lobe lesions. A description of temporal tumours comes with the authority of Prof. Marburg. As for eighth nerve symptomatology, it is examined in relation to a series of toxi-infective states ranging from epidemic encephalitis to morphinism of which it may (or, for that matter, may not) be one of the revealing signs. The reader will thus see how much of general neurological interest is herein included, and perusal

of the above-mentioned chapters will convince him of the high standard of attainment which the writers have set themselves.

As an encyclopædia of otological neurology these volumes form a desirable addition to the specialist's library and we congratulate the editorial staff on the successful accomplishment of their task ; they have produced a remarkable book which is sure to become a ' classic.'

Beiträge zur mikroskopischen Anatomie des vegetativen Nervensystems. By Prof. Dr. PHILIPP STOHR, Jr., Bonn. (Fortschritte der Naturwissenschaftlichen Forschung, ed. by Prof. Dr. EMIL ABERHALDEN : N.F., Hft. 9). With 61 illustrations. Berlin and Vienna : Urban and Schwarzenberg. 1929. Pp. 106. Price Mk. 10.

SINCE the sympathetic system has attracted and continues to attract so much attention in recent years on the part of neurologist and experimental physiologist exact knowledge of its morphological anatomy is a plain desideratum. Numerous statements, current in textbooks and accepted without question, are shown by Prof. Stöhr to require revision, and some of these may here be indicated.

It is very difficult to say where the sympathetic system begins and ends, and impossible microscopically to separate sympathetic from cerebrospinal fibres. Non-myelinated fibres occur among the latter just as definitely as among the former. The distinctions usually relied on as between sympathetic and parasympathetic systems are purely physiological, and certainly not histological. Since sympathetic fibrils end within the actual protoplasm of, for example, smooth muscle-fibre cells, it is altogether impracticable, and anatomically unjustifiable, to claim that a given poison acts on ' nerve-endings ' as distinct from ' cell-substance ' or vice versa. Again, the common separation of myelinated from unmyelinated fibres is proved histologically to have no basis in fact ; it is impossible in numerous instances to determine whether a given fibre is or is not myelinated ; and Prof. Stöhr prefers to speak of fibres poorly or richly myelinated, respectively. Fibres declared to be unmyelinated are in fact surrounded by a fine plasmatic sheath.

There is no individuality in sympathetic nerve fibre-bundles : exchange from one bundle to another constantly occurs ; and it is of peculiar interest to note that though such bifurcations and communications are frequent the actual trunk that forks does not of necessity become smaller in dimensions. Fibres never pursue the shortest course to a given tissue, but are extraordinarily wavy, running through plexuses in a most complicated fashion. No ' free endings ' of sympathetic fibres are ever demonstrable ; rather is there a rete nervosum, a syncytium, at their terminations in cell or other tissues, whereby neural and cytological protoplasm is in practical continuity.

Sympathetic ganglion cells are nearly always polygonal or multipolar, rarely bipolar, and while generically alike, are individually diverse ; no one

cell is ever the exact counterpart of another. The processes of such cells range in number from 3 to 20, more or less; and morphologically no distinction whatever can be drawn between dendrites and neurites, hence it is often impossible to determine which are which. Again, none of these processes end 'free.' In the main paravertebral sympathetic chain the 'white' and 'grey' rami communicantes are by no means so distinct as the epithets suggest, and those of efferent cannot be separated from others of afferent function on anatomical grounds. It is highly questionable whether any sympathetic neurones occur as individual units; on the contrary, the evidence here adduced goes to prove that the whole system is a cellular-fibrillar network, embedded in a protoplasmic continuum.

Much more of lively interest will be found in this excellent monograph, which reveals almost on every page the critical and cautious scientific mind of its author.

S. A. K. W.

Cerebrospinal Fluid in Health and Disease. By ABRAHAM LEVINSON, B.S., M.D. 3rd Edition, with 88 illustrations. London: Henry Kimpton, 1929. Pp. 386. Price 35s.

THE present edition of this well-known book has been expanded by about half its original bulk. This increase in size is chiefly due to an enlargement of the chapter dealing with the changes in "the cerebrospinal fluid in various diseases," and to the addition of a chapter on "the cerebrospinal fluid in infants and children." In both of these chapters little more than a bald statement of the usual changes is given, and with some of these statements, as for example that "in rabies the cerebrospinal fluid is normal," we cannot agree. Little attempt is made to explain the various abnormalities which appear in various diseases: indeed, they all seem to be attributed to a general "irritation of the meninges." Perhaps our knowledge has not yet advanced to the point where we can give a reason for each departure from the normal, but it would certainly have been helpful if a rather more determined attempt to do this had been made. Expansion and rearrangement of the subject matter are noticeable also throughout the book, which has been brought more up-to-date in several directions, notably in regard to technique. Here again, we find that in respect of some important methods of examination the description given is not sufficiently detailed. It would not be possible, for example, for any one who did not possess previous knowledge of the test to perform the Kahn reaction on the cerebrospinal fluid from the directions given in this book. In these respects we feel that there is room for still further improvement in subsequent editions, but the additions which have been made to the book in this edition have considerably enhanced its value.

J. G. G.

Ueber Umbau und Abbau der Sprache bei Geistesstörung. By Dr. F. G. STOCKERT, University of Halle. Berlin: S. Karger. 1929. Pp. 82. Price Mk. 7.80.

A THOUGHTFUL and readable essay in speech pathology is concerned not so much with aphasia sensu strictiori as with disorders of intonation, accent, 'melody,' and rhythm on the one hand, and with mutism, perseveration, verbigeration, 'Ideenflucht,' and speech medley on the other. These various departures from normality are discussed as they occur in a setting or background of mental impairment, and illustrated in an interesting fashion. Reference is made to still other anomalies of both neurological and psychiatric importance, such as the echolalia and coprolalia of Gilles de la Tourette's disease, pleonasms, speech 'contaminations' and 'word-monsters,' etc., such as are encountered in some of the psychoses. The general interpretation of these abnormalities here offered follows in part lines laid down by Wernicke and Kleist, and their relation to certain apraxic syndromes (ideomotor parapraxia) is discussed.

Die Encephalitis Lethargica: ihre Nachkrankheiten und ihre Behandlung. By Prof. Dr. C. v. ECONOMO, Vienna. With 21 illustrations. Berlin and Vienna: Urban and Schwarzenberg. 1929. Pp. 251. Price Mk. 20; bound, Mk. 22.

IN the convenient form of a comprehensive monograph the reader will find everything that is at present known of epidemic encephalitis summarised and critically discussed by the Viennese authority, Prof. von Economo. Where all is so well done it is perhaps of minor importance to comment adversely, yet we cannot refrain from remarking that the least satisfactory aspect of the book is the sketchiness of the attempt to correlate semiology and pathological anatomy. The lacunæ here are really rather striking; and this is the more regrettable since the author is evidently aware of the discrepancies between these two that have characterised a number of reported cases. On the other hand, the richness of the clinical and pathological descriptions separately leaves nothing to be desired. In an endeavour to assign an exact nosological position to the affection comparison and contrast with other varieties of encephalitis are carefully assessed, yet we miss allusion to rabies, which of them all most closely resembles the epidemic disease, especially in its early attack on the substantia nigra.

S. A. K. W.

Der Kopfschmerz und seine Behandlung. By Doz. Dr. EUGEN POLLAK, Vienna. Leipzig and Vienna: Franz Deuticke. 1929. Pp. 158. Price Mk. 9.

HEADACHE, head sensations and neuralgia, beset the human race. Some persons are scarcely ever free from headache, others have never had one in

their lives. A reputedly 'minor' symptom, it is nevertheless accountable for much interference with working capacity as well as for ill-temper and other derivatives that need not be particularised. Dr. Pollak deals compendiously with headache as it appears in association with recognisable causes—infections, toxicoses, vascular disorders, neuroses, and affections of eye, nose, ear, teeth, and so forth. If there is nothing conspicuously novel in his handling of so wide-ranging a topic, headache itself is old and we suppose everything has already been said of it that can be said. He accepts the vasomotor theory of migraine and evidently believes there is some ground for Spitzer's hypothesis of a temporary unilateral hydrocephalic state accompanying its manifestations.

Of considerable practical value are the very numerous therapeutic suggestions to be found throughout the book.

Sulla Sclerosi Laterale Amiotrofica. By Dr. PAOLO OTTONELLO, Pavia. (Vol. xviii of Rassegna di Studi Psichiatrici, ed. by Dr. D'Ormea). Siena: Stab. Tip. S. Bernardino. 1929. Pp. 326. Price not stated.

DR. OTTONELLO has written a fine monograph on Charcot's disease, based on clinical and pathological study of abundant personal material. It is a model of what such contributions should be. As an appendix are provided resumés of his conclusions in French, German, and English—no doubt of value to the hurried reader but, we trust, superfluous for the educated neurologist. His researches show that amyotrophic lateral sclerosis should be considered a systematised affection implicating upper and lower motor neuronic systems independently and often unequally, and they cast doubt on the theory of transsynaptic degeneration. He believes on pathological grounds that the condition is in essence one of primary degeneration and that evidence favouring a toxi-infective hypothesis is scanty and probably negligible.

Oto-Laryngologie, avec Applications à la Neurologie. By F. J. COLLET. Professor of Oto-laryngology in the Faculty of Medicine of Lyons. With 294 illustrations and 4 plates in colour. Paris: Gaston Doin et Cie. 1929. Pp. 1110. Price fr. 170.

PROFESSOR COLLET'S textbook is conceived mainly as an aid to the practice of otolaryngology and the major portion of its thousand pages is concerned with semiology and treatment; what interests the neurologist are the sections devoted to intracranial complications of ear and nose affections, and to various syndromes of palatal, pharyngeal, and laryngeal nerves. As befits its object, the style of writing is somewhat dogmatic and little or no attempt is made to offer complete accounts of differential diagnosis such as would interest the nerve specialist: for example, the palatal palsies of myasthenia are not commented on, palatal nystagmus is given a mere allusion, and while a good description is furnished of the syndromes of Avellis, Jackson, and Tapia the reader is not vouchsafed any account of the circumstances in which these are

likely to be met—at least, not such as would naturally be expected in a neurological treatise. The paragraphs on modern technique in respect of labyrinthine testing are more satisfactory, as are those dealing with disorders of the nervus acousticus and pontocerebellar tumours. Prof. Collet does not provide bibliographical references, but there is a good index—rather a rarity in productions from the French schools.

Anatomical Studies of the Central Nervous Systems of Dogs without Forebrain or Cerebellum. By L. Raymond Morrison, M.D. Haarlem: De Erven F. Bohn. 1929. Pp. 109.

The material on which Dr. Morrison's anatomical studies are founded consisted of the brains of two dogs: one lived 136 days without cerebellum, 83 days without cerebellum, right hemisphere, and right striatum, and 27 days without cerebellum and forebrain (including striatum) on either side. The other animal lived 97 days without left hemisphere, 77 days without right hemisphere, and 38 days without cerebellum. Examination was conducted by serial sections of the formalin-fixed nervous tissues. The many points of anatomical interest established by this research cannot conveniently be summarised, and the original should be consulted by those who desire information. A large number of drawings and photographs reproductions illustrate the author's findings, which bear mainly on pallial connexions with lower centres.

Ueber einige neuere diagnostische und therapeutische Verfahren in der Neurologie. By Dr. Eugen von Thurzó, Debreczen, Hungary. With 11 illustrations. Berlin: S. Karger. 1929. Pp. 78. Price Mk. 10.

Several useful papers on neurological subjects are included in this brochure, of which perhaps that on the question of naturally acquired malaria in syphilitic subjects is the most interesting. The author has had occasion to observe five cases of general paralysis and four of tabes, in each of which the patient had suffered from malaria (tertian or malignant tropical) either during secondaries or a subsequent latent period, yet in none of them did this severe infection prevent the eventual development of metalues. On the other hand, it is stated that the clinical course of these particular cases turned out to be of rather a mild character. In another article the case is described of an eleven-year-old girl suffering from an epileptic psychosis, who for a period of about two months exhibited the phenomena of 'palilogy' and of paligraphy. By the former of these terms the author signifies a condition of what in England would be called iteration or recurring utterance; the child repeated, for example, the words "Ich fürchte mich hier zu bleiben" 10 times, and "Fürchte mich" 28 times, etc. Another case reported in some detail was one of postencephalitic oculogyric crises, taking the form of "à bascule," and seemingly relieved to a considerable extent by suboccipital puncture. Compulsive weeping is mentioned as an apparently new postencephalitic symptom allied to the 'kladzomania' of Benedek.

Studien über Kretinismus. By Dr. ARNOLD FLINKER. With an introduction by Prof. Dr. J. WAGNER-JAUREGG. Leipzig and Vienna: Franz Deuticke. 1929. Pp. 122. Price Mk. 10.

A BEAUTIFULLY illustrated study of the cretins of the Bukowina by the late Dr. Arnold Flinker is noteworthy for its patient gleaning of morphological data, based on over 120 examinations, as well as for a critical correlation of the condition as it appears in Austrian and Roumanian valleys with the geographical features of the country. Dr. Flinker enlarges on the association of goitre with cretinism but is careful to point out that each may occur without the other. He is convinced of a connexion between the disease and certain streams (from which drinking water is obtained) in the regions investigated, yet is unable to find any etiological solution. Among his 120 cases we note an occasional reference to nervous symptoms such as weak legs and club-foot, but no description is specifically given of so-called nervous cretinism.

Études Cliniques sur les Syndromes Moteurs et Réflexes de la Paroi Abdominale et d'Origine Cérébrale. By Dr. AKE BARKMAN. Uppsala: Almqvist and Wiksell. 1929. Pp. 304. Price not stated.

MANY years ago Professor Pierre Marie of Paris was wont to say that hemiplegia is as valuable as a physiological experiment and that more of cerebral physiology could be learned by its study than from any other morbid cerebral condition. Curiously enough, however, little new study has been made of its inviting semiology, and we therefore welcome Dr. Barkman's advances in respect of trunk disorders produced by cerebral lesions, the more so because he mentions data and reaches conclusions with which the reviewer himself has long been familiar and which he has long taught. Briefly, Dr. Barkman is satisfied that Broadbent's law requires revision, and that unilateral involvement of abdominal musculature is the rule and not the exception in recent hemiplegia. Minute as his clinical researches are, we do not think they cover all the facts, nor has he always been conversant with foreign work on the same subject, since there seems to be no allusion to the late Dr. Beevor's Croonian Lectures of 1904. But the monograph is nevertheless of considerable interest.

Die Lehre von den Neuronen und vom Neurencytium und ihr heutiger Stand. By Prof. Dr. HANS HELD. Leipzig. (Fortschritte der Naturwissenschaftlichen Forschung, ed. by Prof. Dr. EMIL ABDERHALDEN: N.F., Hft. 8). With 21 illustrations. Berlin and Leipzig: Urban and Schwarzenberg. 1929. Pp. 44. Price Mk. 5.

IN a clearly written essay the neurologist will find a concise review of the neurone doctrine, a historical survey being followed by a critique of present-day theory and a description of recent advances in histological knowledge. Neurones as genetic and anatomical units have no existence; the neuroblasts of the embryo develop along plasmatic paths exclusively in an encytial fashion. The nervous

system cannot be defined as a summation of neurones ; it is a syncytial network throughout. It is constituted by neurocytes, whose fibrillæ pass over more than one, or an actual series, of 'neurones.' The unity of the system is based on this fibrillar continuity.

Acute Infectious Diseases : a Handbook for Practitioners and Students. By J. D. Rolleston, M.D., M.R.C.P., London. Second edition. London : Heinemann (Medical Books) Ltd., 1929. Pp. 419. Price 15s. net.

Among the chapters of direct neurological interest in Dr. Rolleston's thoroughly modern and eminently informative textbook are those concerned with diphtheria and cerebrospinal fever, while the nervous side of the commoner eruptive fevers (such as measles, scarlatina, typhoid, varicella and smallpox) and of other infections such as whooping-cough and vaccinia, receives ample consideration. In view of present-day attention to infections of the nervous system a book of this kind, written from the standpoint of the fever expert who is also conversant with nervous disease, with useful lists of references, is of real value to the professing neurologist and as such can be warmly recommended to our readers.

Prescribing Occupational Therapy. By William Rush Dunton, Jr. London : Baillière, Tindall and Cox. 1929. Pp. 142. Price 10s.

This little book gives a concise and clear account of the uses of occupational therapy in various kinds of illness—mental, orthopædic, cardiac, etc. In the mental field the author indicates briefly how a given patient may be helped more by a particular type of occupation than by any other, if the prescribing physician knows sufficiently how to make use of the resources both of the patient and of the hospital. It is obvious that for the proper utilisation of occupational therapy a special provision and organisation of teachers and accommodation in hospital is necessary. In the section on mental diseases, the author is rather too much impressed with the importance of avoiding fatigue in various psychoneurotic conditions : it would be more rational to impress most of such patients with their emotionally sound physical and mental capacity.

The book contains useful lists of references and will serve as a convenient and succinctly practical guide to the subject.

Endocrine Disorders. By Prof. Hans Curschmann, Director of the Medical Clinic, University of Rostock. With an introduction by Dr. F. Prange, Assistant at the Clinic. London : H. Milford, Oxford University Press. 1929. Pp. 188. Price 12s. 6d. net.

Written essentially from the standpoint of the practising clinician, Professor Curschmann's slender but concise and comprehensive volume can be cordially recommended on its merits. Anatomy and physiology are mentioned by way

of brief introduction in respect of each of the ductless glands, and succeeded by well illustrated descriptions of the outstanding and least controversial syndromes singly and in combination. So far as we have been able to note the ground is covered with some pretence to completeness: the progeria of Gilford, however, is not alluded to, nor the retrograde infantilism of Gandy, although their symptomatology receives attention incidentally along other lines. Certain forms of dwarfism are annotated whose connexion with the endocrine system is dubious, but after all it is clinically convenient to make an arrangement of this kind. Disorders of the gonads are well discussed, with short accounts of hermaphroditism, sexual inversion, homosexuality, and rejuvenation. A brief appendix deals sketchily with certain conditions (myasthenia, myopathy, sclerodermia, osteomalacia, etc.) considered by some to have an endocrine basis.

Unpretentious in style and yet eminently clear and readable, this little book provides an excellent introduction to a peculiarly complicated subject.

Arbeiten aus dem Neurologischen Institute an der Wiener Universität. Edited by Prof. Dr. Otto Marburg. Vol. xxxi. Leipzig and Vienna: Franz Deuticke. 1929. Pp. 192. Price Mk. 40.

The latest volume in this well-known series contains a number of papers of neurological importance, mainly by Japanese workers. Dr. Nishii writes on ganglion-cell inclusions in epidemic encephalitis—the 'minute bodies' of Da Fano and Ingleby. An elaborate research by Dr. Deutsch bears on alterations in the corpus geniculatum externum as a sequel to tabetic optic atrophy and throws light on the intimate structure and neural relationships of that body. An interesting case of senile disseminated sclerosis (onset at age of 63, death at 74), with confirmation at necropsy, is reported by Dr. Taga. Changes in the spinal cord are shown by Dr. Kurosawa to accompany osteomalacia. Postencephalitic disorders of respiration seem to be associated, in part at least, with lesions of the dorsal section of the vagal nuclear region, according to Dr. Hazama. A long histopathological study of the choroid plexus of the lateral ventricles, in a number of widely differing morbid states, is contributed by Dr. Auersperg.